[October, 1914.]

BRAIN.

PART II., VOL. 37.

THE PHENOMENON OF "TONIC INNERVATION" AND ITS RELATION TO MOTOR APRAXIA.[1]

BY S. A. KINNIER WILSON, M.D., B.Sc., F.R.C.P.,

AND

F. M. R. WALSHE, M.D., B.Sc., M.R.C.P.

Introduction.
Personally observed Cases.
Considerations suggested by the Analysis of our Cases.
Review of Analogous Cases from the Literature.
Terminology and Relation to Perseveration, properly so called.
Nature of the Phenomenon and Relation to Apraxia.
Relation to the Pyramidal System.
Relation to Myotonia, properly so called.
Relation to certain Involuntary Movements.
Pathogenesis and Localizing Value.
Bibliography.

In a paper by Wilson [42] entitled, "A Contribution to the Study of Apraxia," which was published in Brain in 1908, reference was made to the phenomenon of so-called "tonic perseveration," and a personally observed case was briefly alluded to. The details of that case are here published for the first time. Since that date, we have had the opportunity of studying two further instances of the condition, and this paper is therefore based on an investigation of three cases in which the phenomenon was present in a typical and advanced form, constituting, indeed, the most marked clinical feature. We are greatly indebted to Dr. H. H. Tooth, of the National Hospital, Queen Square, London, under whose care the latter two patients were, for permission to record them. Two of these three patients were successfully operated on for cerebral tumour; hence we are in a position to discuss the localizing

[1] A brief communication on this subject was made before the Neurological Section of the International Congress of Medicine, London, August, 1913.

value of the symptom, which is one of considerable physiological interest.

It must be remarked at the outset that the terminology of the condition is far from satisfactory. No rigorous definition of the expression "tonic perseveration" has hitherto been proposed, but it is understood to signify inability, owing to a central lesion, to relax a given innervation in any muscular group or groups. To give a single illustrative example: Liepmann [27], who coined the term, uses it of his patient the *Regierungsrat*, who was occasionally observed to be unable to let go an object which he held in his left (dyspraxic) hand, grasping it more firmly instead. The innervation of the muscular groups concerned seemed to persist in spite of the patient's endeavour to inhibit it. He could not cut short a muscular contraction once it was begun. Liepmann proposed to denote this type of perseveration "tonic perseveration." We prefer, however, the term "tonic innervation," for reasons which will subsequently appear, as being more accurate and less confusing.

As a matter of fact, however, the terminology not only of this, but of the other two varieties of perseveration distinguished by Liepmann, viz., "clonic perseveration" and "intentional perseveration," is open to obvious objections, which will be mentioned when the matter is discussed in the second half of this paper. It is curious that as far as can be discovered from a careful examination of the passages in his various papers bearing on perseveration Liepmann nowhere makes any allusion to the familiar symptom of myotonus or myotonia in connexion with his "tonic perseveration," nor is the word so much as mentioned by Kleist [23] as far as we have noticed, in his latest contribution to the subject of disturbances of psychomotor function. This appears the more remarkable as, clinically, there is no very obvious difference between the inability to relax a handshake of the patient with myotonia atrophica, that of a patient with Thomsen's disease (myotonia congenita), and that of a patient with "tonic innervation." It is, however, to be clearly understood that in tonic innervation we are supposed to be dealing with a phenomenon of central origin, whatever be the site of the lesion in the other above-mentioned diseases.

This brief allusion, then, to what the term is intended to signify, is simply by way of introduction to our three cases. The many questions of interest suggested by a consideration of them will thereafter be approached.

Case 1.—A. J., male, aged 38, was admitted to the National Hospital under the care of Sir William Gowers on December 16, 1907.

The patient had been married seventeen years: his wife had had three stillborn children, but no miscarriages. He was a right-handed man. He denied venereal disease, but admitted risk.

History of present illness.—In February, 1907, he had an attack of influenza, accompanied by a copious thin discharge from the left ear. About a month later, when playing the piccolo at a public concert, he suddenly felt giddy, began to tremble, and had to stop playing. A few days later, he noticed that he was beginning to drag his left leg as he walked. The onset was quite gradual, and involved the whole of the limb. About one month later, the calf of the left leg used to become cramped as he lay in bed, and involuntary flexor spasms of the limb occurred. About the same time when performing dumb-bell exercises he found he could not make rotatory movements of the left arm easily, and also that he sometimes could not relax his grasp of the dumb-bell in his left hand for a second or two. He noticed the same difficulty in relaxing his fingers when he held a fork in his left hand, also in buttoning and unbuttoning his clothes. There were no cramps in the left arm or hand, and no paræsthesiæ in the limbs. The left arm gradually became weak. For two months prior to admission he had suffered from nausea, without actual vomiting. For a month he had noticed occasional involuntary twitches of the left arm, the limb being abruptly elevated so that the elbow was almost on the same level as the shoulder, while some flexion at the elbow occurred: with these movements some slight twitching of the leg was usually observed. The total duration of these little attacks was a few seconds, and they took place several times a day. During the same month he had been conscious of failing vision, more particularly in the left eye.

State on admission.—The patient was seen to be a heavy, stout, florid man, whose intelligence and attention were good, while his memory was poor. In the right eye the vision was $\frac{6}{9}$, in the left $\frac{6}{12}$. There was advanced double optic neuritis, with 6 dioptres of swelling on the right, and 8 on the left. The pupils were unequal: the right small, irregular in outline, and inactive to light. The left was slightly larger, less irregular, and reacted to light sluggishly. The left pupil moved better on accommodation than the right. There was no ptosis, diplopia, strabismus or nystagmus. The left side of the face was slightly weak in its lower part.

Motor system.—The patient's general muscular development was excellent. There was a certain increase of tonus in the limbs on the left side. There was slight general weakness of the left arm compared with the right, and of the left leg compared with the right, most noticeable in the distal segments. The remarkable feature about the patient's condition was his inability to relax the muscles of the left arm (the phenomenon was less pronounced in the muscles of the leg) after they had been employed in muscular effort. If an object was firmly grasped in the left hand, for a few seconds, the patient was quite unable to relax his grasp, for as long as fifteen seconds, or more, even though he made powerful associated movements with the right hand. If he extended the left arm at the elbow, he was unable to flex it again till some five or eight seconds had elapsed. Direct muscular excitability was not obviously increased.

There was no change in sensibility to the usual tests. The abdominal reflex on the left side was diminished: a double flexor response was obtained. The deep reflexes in the legs were equal: in the arms, the left were brisker than the right.

January 21, 1908.—With the right hand the patient used correctly the following objects: Paper-knife, key, scissors, nail.

Movements on request.

With the right hand:	
Turn the handle of a barrel organ.	+
Count out money.	+
Movement of shaving.	+
With the left hand:	
Turn the handle of a barrel organ.	Stiff, rather angular rotation movements of whole arm from shoulder.
Smoke a pipe.	+
Button jacket.	Made fair attempt, the fingers being moved very stiffly and slowly.
Put left hand on top of head.	+
Put left forefinger on tip of nose.	+
Put left forefinger into right ear.	Put it into left ear.
Touch tips of left fingers with left thumb.	At first failed because of inability to relax the necessary innervations, but succeeded somewhat better at a second attempt.
With both hands:	
Make the movements of playing the flute.	The attitude of the two hands correct: movements of fingers correct on right side, very stiff on left, but approximately correct.

There was no agraphia or dysgraphia with the right hand. Very good attempts to draw a pipe, a spade, a Union Jack, were made. In writing or drawing with the left hand, it was obvious that the difficulty was entirely due to the patient's inability to move his left arm with any freedom, the fingers and hand frequently became immobilized for some seconds, and he constantly used the right hand to help the other by taking hold of it and moving it about.

February 3, 1908.—The weakness of the left side of the face was now more marked. A special examination was made of the "tonic innervation" phenomenon.

(1) Strong grasp with left hand for 5 seconds.
(2) Relaxation took 20 seconds.
(3) Flexors were then faradized for 20 seconds.
(4) Then grasp, 5 seconds.
(5) Again relaxation took 20 seconds.

Interval of 2 minutes.

(1) The arm was extended strongly for 5 seconds.
(2) It took 35 seconds for the triceps to relax.
(3) The triceps was then faradized for 20 seconds.
(4) Arm extended as before, 5 seconds.
(5) Relaxation did not occur, in spite of every effort on the part of the patient, for about 38 seconds.

With another trial of the same nature, 105 seconds elapsed before the triceps was relaxed.

On several occasions, when the patient was grasping the fingers of the examiner and was then told to relax, the grasp actually became tighter, and this in spite of the fact that the antagonist extensors were both seen and felt to be contracting in the effort to overcome the tonic innervation of the flexors. The phenomenon was clearly not due to defective innervation of the antagonists of the contracting group.

In the left leg the same feature was present, but in less degree; a succession of quick alternating movements at knee or at ankle was quite impossible because of the difficulty in relaxing a muscle once it had contracted.

The patient's gait was now definitely hemiplegic, but it was noticeable that he was able to move the left leg better when walking than might have been supposed.

The deep reflexes were distinctly brisker on the left side than on the right, there was a tendency to ankle-clonus on the left, and the left plantar response was not so definitely flexor as the right. While the patient was in hospital no movements of the "pseudo-spontaneous" type were ever observed.

February 10, 1908.—The patient was re-examined with regard to the possible effect of massage on the phenomenon of tonic innervation.

(1) Left hand and arm had been lying at rest for some minutes. Grasp for 5 seconds, strongly.
(2) This took 8 seconds to relax.
(3) The extensors of wrist and fingers were then massaged for 2 minutes.
(4) Grasp, 5 seconds' duration, as before.
(5) This was instantaneously relaxed.

After an interval of about 2 minutes, the arm was tested again.

(1) Grasp, 5 seconds.
(2) This took 10 seconds to relax.
(3) The extensors of wrist and fingers were massaged for 2 minutes.
(4) Grasp for 5 seconds, as before.
(5) This took 15 seconds to relax.

Interval of 5 minutes.

(1) Grasp, 5 seconds.
(2) This was relaxed instantaneously and easily.
(3) Massage to the extensors for 2 minutes.
(4) Grasp for 5 seconds.
(5) This took 75 seconds to relax.

The patient was tested again, some hours later.

(1) Grasp, 5 seconds; this was relaxed in 5 seconds.

(2) Grasp, 5 seconds; this took 10 seconds to relax.

(3) Massage to extensors, 2 minutes; grasp, 5 seconds; relaxation took 40 seconds.

Massage in the following tests was given to the flexors of wrist and fingers.

(1) Grasp, 5 seconds; this was relaxed in 10 seconds.

(2) Massage to flexors; grasp, 5 seconds; relaxation, 10 seconds.

The experiment was repeated.

(1) Grasp, 5 seconds; relaxation, 10 seconds.

(2) Massage to flexors for 2 minutes; grasp, 5 seconds; relaxation, 2 seconds.

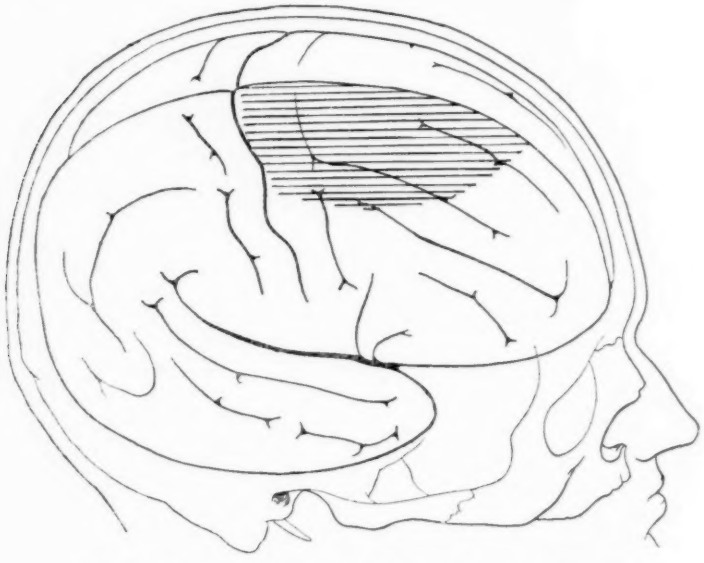

FIG. 1.—Shaded area indicates site of tumour removed in Case 1.

February 11, 1908.—Sir Victor Horsley operated under chloroform, making a large craniectomy over the right frontoparietal region. The dura was seen to be very tense, but not discoloured. It was not opened at this stage. The patient stood the operation very well.

February 18, 1908.—Under chloroform the wound was reopened and the dura incised freely. It was at once seen that the convolutions were much flattened and the brain looked pale. A horizontal incision was made in the posterior end of the superior frontal gyrus; about ¼ in. below the surface a large dark plum-coloured encapsulated mass was discovered. After ligaturing certain

branches of the anterior cerebral artery, the operator explored with his finger round the tumour and was able to remove in its entirety a large endothelioma, encapsulated above but somewhat broken on its under surface, about the size of a tangerine orange. The tumour had extended backwards under the upper third of the precentral gyrus: it extended forwards well into the frontal centrum ovale, and bulged over the midline, pressing on the right side and upper aspect of the genu of the corpus callosum. The lateral ventricle was not opened. The patient stood the operation well.

February 21, 1908.—After the operation there was complete flaccid hemiplegia of the left arm and leg, while the face was less involved. Nevertheless on passive movement at the elbow, flexion and extension alternately, there was always some momentary resistance in the antagonist. This was also found when alternating passive movements were made at the knee. An extensor response was obtained on the left side. The phenomenon of tonic innervation, however, had completely disappeared.

March 24, 1908.—By this date the patient's general health had greatly improved. There was some return of power in the flexors of the left elbow, wrist and fingers, but otherwise the hemiplegia was as before. Slight relative anæsthesia and hypalgesia were present over the left side of the body. An extensor response was obtained on the left and a flexor on the right. There was no reappearance of the tonic innervation.

Five years later (February 11, 1913) the patient reported himself as being in excellent general health, although the hemiplegia was practically unchanged.

Case 2.—W. L., male, aged 47, was admitted to the National Hospital under the care of Dr. H. H. Tooth in May, and again in August, 1912.

The patient had always been a healthy man, was right-handed, and denied venereal disease.

History of present illness.—Twelve months before admission, while he was lying down on a couch at home, his left foot suddenly began to jerk. He was quite conscious and suffered no pain, nor was there any subjective sensation. The jerking spread rapidly up the leg, then appeared at the shoulder and spread down the arm to the hand. The whole fit lasted about ten minutes. Thereafter he noticed that the left arm and leg were weak, and this weakness gradually progressed, though there were no more fits, till for the last four months he dragged the leg very badly when walking. He did not complain of headache, vomiting, or any affection of vision.

State on admission.—The patient was seen to be a well-nourished plethoric-looking individual. His intelligence, memory and attention were good. There was early optic neuritis in both eyes (+4D. R. and L.) with acuity of vision $\frac{6}{9}$ R. and L. The facial lines on the left side were slightly flattened, but the tongue was protruded straight. There was slight spastic paresis of the whole of the left arm, most marked in the distal joints. A similar condition obtained in the left leg. Slight relative hypalgesia and hypæsthesia were present over the left side of the body, including the limbs. The tendon reflexes were brisker on the left than on the right, and an extensor plantar response

was obtained on the left side. The Wassermann reaction in the blood was negative.

The patient remained for about one month in hospital, and went home with his condition unchanged.

Some two months later he was readmitted with an aggravation of symptoms. The weakness on the left side had steadily increased, and in addition he complained of failing vision.

On re-examination the optic neuritis was found to have progressed in both eyes, vision now being $\frac{6}{12}$. There was a minor degree of weakness of the left external rectus. The lower facial weakness on the left side, previously noted, was still present. The hemiparesis of the left limbs was distinctly worse than on the previous admission, and the spasticity had increased in degree. The left arm assumed the typical hemiplegic attitude of flexion and adduction. There was still, however, considerable power and range of movement.

After a stuporose attack on September 5, it was observed that the phenomenon of tonic innervation was present in a striking degree in the left upper limb; it was not noted, however, in the left leg. Owing to the awkwardness with which the patient passed from one movement to another it was difficult to test for apraxia. Such tests as could be performed revealed no definite dyspraxia, hence only a brief selection need here be given.

	RIGHT.	LEFT.
Asked to beckon:	+	Recognizable attempt.
To button his shirt:	+	do.
Shake his fist:	+	Slow but good attempt.
Strike an imaginary match (L hand) on an imaginary box (R hand).		Recognizable attempt.
Sharpen a pencil with knife in L hand.		Instead of moving knife against pencil he moved pencil (R) against knife held immobile L.

Examination of the phenomenon of tonic innervation.— When asked to place his left hand on his head he did so readily. It was allowed to remain there five seconds, and he was then requested to bring it down to his side again. This took no less than three minutes, and even then was not completely accomplished; however, annoyed at his helplessness, he pushed the arm down with his right hand.

(1) Grasp left hand, 5 seconds.

(2) This took no less than $2\frac{1}{2}$ minutes to relax.

Faradization both of flexors and of extensors for 30 seconds made no difference in this relaxation.

Effect of massage.

(1) Grasp, 5 seconds (the extensors having been previously massaged).

(2) Relaxation in 2 seconds.

(3) Grasp repeated one minute later; relaxation without further massage, 35 seconds.

(4) Repeated 10 minutes later, relaxation 2½ minutes.

The flexors having been previously massaged:—

(1) Grasp, 5 seconds.

(2) Relaxation, 5 seconds.

(3) Grasp repeated one minute later; relaxation 5 seconds.

(4) Repeated 10 minutes later; relaxation 2½ minutes.

In endeavouring to relax his grasp it was clear that the patient did try his best to accomplish his object, and understood perfectly what was required. If the examiner attempted to withdraw his fingers rapidly from his hand the grasp of the patient's fingers always tightened perceptibly. On clenching his

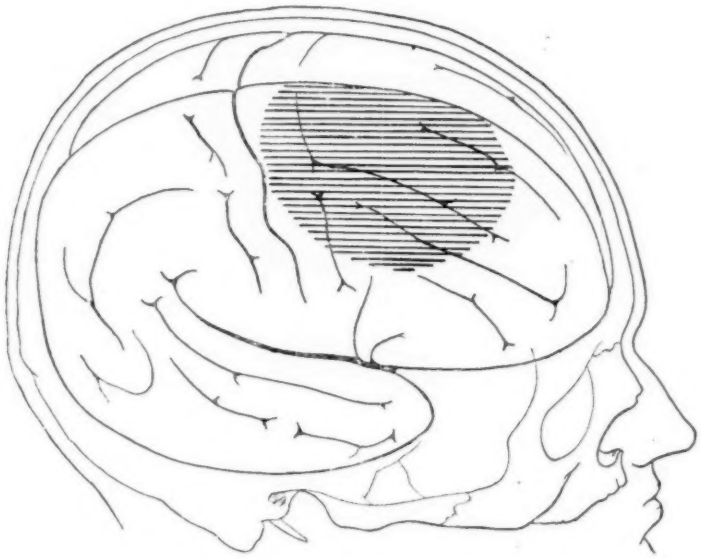

FIG. 2.—Shaded area indicates site of tumour removed in Case 2.

empty fist he seemed to have less difficulty in relaxing—35 seconds usually. He was asked to flex his arm forcibly against resistance, which was maintained for 5 seconds. He was then requested to extend his arm: this took 50 to 60 seconds. During this time the contraction of the biceps slowly died away (as felt by palpation), while the triceps came at once into action and only predominated as the contraction of the biceps passed off.

The completeness of the patient's comprehension of all that he was asked to do was assured by asking him to perform the movements with his right hand, or arm; this he always did readily and easily.

Sensation.—On the left side of the body and left limbs, pain, temperature,

and localization were unimpaired, but there was mild hypæsthesia to cotton-wool, and there was a very moderate diminution in the sense of position. There was no astereognosis.

September 6, 1912.—Right frontal decompression, first stage. Mr. Sargent removed the bone and opened the dura, exposing a plum-coloured tumour situated in the frontal and prefontal region, as in fig. 2. It was circumscribed and well defined in outline. The patient bore the operation badly.

September 13, 1912.—Second stage: The tumour was exposed and found to be superficial, displacing without actually invading the brain substance. It was removed *in toto*, leaving a bed of depressed cortex. It grew from the falx cerebri by a somewhat pedunculated base, and was of the dimensions of a tangerine orange. It lay exactly over the posterior ends of the first and second right frontal convolutions, extending back to the anterior margin of the fissure of Rolando in its upper part. Its lower border reached $2\frac{1}{2}$ in. laterally from the mid-line. The tumour was an endothelioma.

September 22, 1912.—The hemiplegia became more marked after the operation, for the time being, whereas the phenomenon of tonic innervation completely disappeared.

October 4, 1912.—The patient's general condition was by this date very good. The left upper extremity showed complete paralysis with some spasticity, and the phenomenon of tonic innervation was still quite absent. Eventually a fair recovery was made, and the patient left the hospital in good general health and walking well.

Case 3.—F. F., female, aged 58, was admitted to the National Hospital under the care of Dr. H. H. Tooth on September 5, 1912.

Previous history.—The patient was a right-handed woman who had always enjoyed good health till within a few years of her admission to hospital. For the previous two years or more she had complained of occasional severe "cramps" in the left thigh, and a frequent feeling of numbness and deadness in the left hand, of some minutes' duration. For about the same period of two years the patient had been rather depressed and easily moved to tears.

History of present illness.—On May 15, 1912, she complained of "pins and needles" in the left leg, and by the next morning she had lost the use of the limb. On that day she vomited copiously, and in the course of a few hours the left arm became as "useless" as the leg. There was no loss of consciousness and no headache. Two or three days later she lost all feeling in the left limbs, but this returned in the course of three weeks. Since the onset of the illness there has been a certain amount of mental deterioration; memory has become defective, and the patient has been emotional and unreasonable, and not always, apparently, aware of her failure to control the action of her sphincters. Before admission the left leg improved slightly, but the arm did not. The patient noticed she was unable voluntarily to unclasp the fingers of the left hand, or to let go of things she was holding in the left hand. Occasional brief periods of a degree of dysarthria were noted since the commencement of the illness.

State on admission.—The patient was a healthy-looking florid-complexioned woman, not complaining either of pain or discomfort.

Her attention was not always very good. Her memory for recent events was somewhat defective. During the examination she frequently smiled or laughed for inadequate reasons, and made irrelevant remarks.

Speech.—She comprehended spoken and written speech perfectly. There was no failure in identifying objects and pictures of objects. There was no defect of spoken language. She wrote quite well with the right hand, but there was a tendency, in the first place, to the occasional omission of a letter or a word, and in the second, to a degree of perseveration (in the strict sense of the word, see below). Thus she wrote "God sve th king"; "have yo Pears' soap?" for "have you used Pears' soap?" "Natioal Hospital"; "our Father who art heaven," &c. The perseveration is illustrated by the following: she wrote her name correctly the first time, "Fanny Flynn." The second time she wrote "Fanny Fanny." For "it is a fine day," she wrote "it is a finne day." With the left hand she could do nothing whatever as far as an attempt at writing was concerned.

FIG. 3.—(Case 3.) With the normal right arm patient readily puts finger on nose.

Apraxia and tonic innervation.—As already mentioned, there was a degree of left hemiplegia present. In the case of the left leg there was complete paralysis with contracture. In the arm, however, there was still considerable power present, and no trace of contracture or contraction-attitude. The remarkable feature of the arm condition was, in the first place, the complete motor apraxia. The patient understood perfectly what was required of her, and made no mistakes with her right arm. All the time that the patient was under examination she was seen to move the

left arm freely, and to execute in normal fashion well co-ordinated movements, but only in an automatic way. Thus at one moment she was fingering the bedclothes with the left hand and pulling them up—a favourite occupation—at another, she was scratching her left thigh; then she was seen twisting her wedding-ring, which she wears on her right hand, round her finger with her left forefinger and thumb; in fact, her left arm was practically never still. In striking contrast to this freedom of involuntary movement (a typical example of so-called "pseudo-spontaneous" movement) was her inability to perform any movement with that hand or arm, even the simplest and most elementary, when she was asked to do it, i.e., when on request she consciously endeavoured to use the limb.

FIG. 4.—(Case 3.) With the apraxic left arm patient cannot put finger on nose, and has to grasp the left arm with the right in order to perform the test.

The second remarkable feature of the left arm and hand was the condition of tonic innervation they presented. When the examiner put his fingers into her left hand, her fingers at once closed on his, and she was then unable to relax her grasp. The more he tried to release his fingers the more tightly hers closed round them, until she spontaneously caught hold of his hand with her right and pulled it away from her own left grasp. Once this was accomplished, her left hand relaxed, possibly because she was no longer exercising any conscious volition over it. It was also noticeable that if, while the examiner's hand was still grasped in her left, her attention was successfully diverted, then the examiner could remove his hand more easily. In one of the tests for apraxia a hair-brush was put into her left hand and she was quite unable to let it go when she wanted, and had to make great efforts to pull it away with her right hand.

Some examples may now be given of the phenomena observed during examination :—

Right hand :

Beckoning.	+
Shake fist.	+
Shake hands.	+
Open hand.	+
Close hand.	+
Put hand on head.	+
Touch thumb and little finger.	+
Make movement of cutting with a pair of scissors.	+

With objects in the right hand :

Brush, comb, pipe, button-hook, mirror.	All correctly and readily used.

It was obvious, in short, that the right limbs were normal in every way.

Left hand.—During examination the left hand was practically never still; the patient usually either held the bedclothes with it, or kept playing with her nightdress with her fingers, or pulled and pushed the bedclothes up and down.

Shake hands.	"I can't quite"; put out three ulnar fingers.
Open your hand.	Repeated the movement of three ulnar fingers.
Close your hand.	Opened it, then caught hold of the bedclothes and pulled them up; "I can't."
Put hand on head.	"I can't, sir"; all the time she moved her hand about at her neck and chin in an aimless way. "It's too heavy"; then seized it with the right hand and tried to put it on to her head.
Touch thumb and each of fingers in succession.	"I can't, sir"; put thumb in between third and fourth fingers and kept it there.
Shake fist.	A fair attempt.
Beckoning.	"Isn't it funny I can't do it? It is certainly very extraordinary." Opened hand and simply looked at it.
Cut with pair of scissors (no object).	Repeated exactly what she did for "beckon."

Tested with objects in the left hand :

Brush.	This was put into her left hand; she grasped it firmly, turned it round and round, did nothing else, and was incapable of letting it go. The brush had to be pulled away by main force. The brush was put into hand again, and then she was invited to "let it drop." Of this she was absolutely incapable. She kept twisting and turning

	it round and round, moving her fingers over it. A moment before, she had opened her hand merely by looking at it, and it was frequently the case that the examiner could get her to relax her grasp by saying *suddenly*, "Look at your hand," when she automatically opened it and looked at her palm. She was not told to *open* her hand, but merely to *look* at it.
Comb.	Holding the comb firmly in an awkward way, she put it into her mouth, and made an irregular movement as though to comb to and fro. Knew quite well that this was wrong, and said so.
Button-hook.	Did nothing with it. Said it was a button-hook, then made wild endeavours to get it out of her left hand and transfer it to the right.
Mirror.	Held it reversed, but managed to turn left forearm round so as to look into the glass, and was able (on this occasion) to let it drop from her hand.
Bilateral movements:	
Put out tongue.	Correct. Kept tongue protruded as though she had forgotten it was out.
Shut your eyes.	"I can't do it": shut them for a single moment only. "You've done it." "Have I? I can't do it." Could not in any way manage to keep them closed for longer. The closure seemed more involuntary than otherwise. Could always close them if an object was approached to them.
Whistle.	Fair attempt, after a failure.
Cough. Wrinkle forehead. Frown: blow out cheeks. Sigh. Take a deep breath.	All done more or less correctly. In the case of wrinkling the forehead, and frowning, the movements were well sustained.
Make movements of playing the piano.	Right correct, left simply nil.
Put your hands as in prayer.	Right instantly put up to correct position, while the left remained entangled in the coverlet of the bed.
Tie a knot in a piece of string (given).	By using her teeth and her right hand mainly she managed to accomplish this. Her left fingers caught in the string and she could not get them out.
Open a pencil-holder (given).	Managed this also, using the right hand almost entirely. She could not get the end of the holder out of her left hand. "Isn't it tiresome?"

	The next moment she had grasped the clothes with her left hand and pulled them up to her chest.
Strike a match on box (given).	She moved the box up and down in an ineffective way against the match head, held in left hand. When the examiner had pulled the match out of the patient's left hand, she suddenly put her left hand up with the greatest readiness and wiped the point of her nose with it.

October 10, 1912.—Both apraxia and tonic innervation were unchanged. The patient's memory was much as before; however, she seemed to be remembering incidents of her hospital life very well. Her attention was still easily distracted. Orientation in time and space was much improved from the time of her admission. There were neither hallucinations nor delusions. Her habits were still faulty, i.e., her control over her sphincters seemed impaired.

FIG. 5.—(Case 3.) In a bilateral test ("hands as in prayer"), normal right arm and hand; left hand and arm, apraxia and tonic innervation (patient cannot let go the bedclothes).

The patient's faculty of imitation of movements was examined. The hands of the observer were placed together in the attitude of prayer, the patient having been previously asked to imitate the movement. Her right hand was immediately placed in the middle line of the body in the desired attitude, while the left hand was slowly and imperfectly brought into a position more or less analogous, the patient having great difficulty in releasing the bedclothes which she was grasping with her left hand. Sometimes she brought the hand up and the bedclothes along with it. It frequently happened that the patient

FIG. 6.—(Case 3). Patient endeavours with her right hand to pull a small object out of the left hand which grasps it firmly. Note innervation of the left extensors in the endeavour to overcome the tonic innervation of the left flexors.

FIG. 7.—(Case 3.) Patient is unable to relax the tonic innervation of her left flexors.

imitated with her right hand the movements that the examiner was making with his left; often only a very feeble attempt was made with the left, apart from the tonic innervation of the limb.

The performance of single movements with the left hand, on request, was as imperfect as on the patient's admission. Movements of expression, such as "throw me a kiss," were correctly performed on the right side; on the left

FIG. 8.—(Case 3.) The more patient tries to inhibit the tonic innervation, the firmer does her grasp become, so that the observer is able to pull her arm right over to the other side.

the hand was very slowly and imperfectly approximated to the lips; the head was bent forward to meet the hand, which remained stiffly in position although the patient then withdrew her head. The tonic innervation remained unchanged.

Examination of rest of nervous system.—No defect of cranial nerves beyond slight weakness of the muscles of the left side of the face was present. The ocular movements were free; there was no nystagmus. The optic discs were normal, the pupils were regular and equal, and their reactions normal. There was definite though slight general weakness of the left arm, and to a greater extent of the left leg. There was some spasticity of the left arm, apart from

the tonic innervation, and more in the leg, which was in a state of contracture.

Examination of the sensory system was difficult because of the patient's failure to attend properly. Apparently there was slight diminution of cutaneous sensory acuity in the peripheral part of the left arm. Muscular sense was probably somewhat diminished distally. At one time it appeared that the patient was not always able to recognize by touch objects held in the left hand, but this condition varied and was not constant. There was no ataxia of the left arm.

The tendon reflexes on the left side were brisker than on the right: there was ankle clonus on both sides, left greater than right, and a plantar extensor was always obtained on the left side, probably a flexor on the right. The Wassermann reaction in the blood was negative.

Before the patient left hospital (December 14, 1912) it was noted that with the eyes closed her control over the movements of the left side was extremely poor. The apraxia and tonic innervation, especially the former, seemed worse when the eyes were closed; if they were open the apraxia improved although she was not actually looking at the way in which the arm was moving.

If, now, we summarize succinctly these three cases, their chief features may be described as follows:—

In Case 1 a male patient, aged 38, gradually developed weakness of the left leg and later of the left arm, and simultaneously with the latter he found that he was unable to relax the muscles of the left arm readily after they had contracted in any voluntary act. During the same time occasional little attacks of involuntary twitching of the musculature of the left arm occurred. Occasionally, also, cramps and involuntary spasmodic movements of the left leg took place, mostly as the patient lay in bed. On examination he presented characteristic signs of cerebral tumour, with headache, nausea, and advanced double optic neuritis. The muscles of the left face and limbs were weaker than those of the right, and there was distinct increase of tonus in them. The left abdominal reflex was diminished, and the tendon reflexes on that side were increased, but there was a double flexor response. No alteration in sensibility could be detected, and there was no ataxia. In the left arm, and to a less extent in the left leg, the phenomenon of tonic innervation was present in a striking degree. If an object was grasped in the left hand the patient was unable to relax the innervation for a full minute or more, although he made powerful associated movements with the right arm, and although the tendons of the antagonist group could be seen distinctly to contract. Frequently the grasp became tighter the more he endeavoured to inhibit it. Other muscular groups,

both flexor and extensor, showed the same phenomenon. The right arm was entirely normal. There was no apraxia or dyspraxia of the left arm, but the defect of innervation hindered the patient in carrying out requests. There were no involuntary movements of the "pseudo-spontaneous" type. Fair attempts at writing were made with the left hand. The left limbs gradually became more hemiplegic, and ankle-clonus developed. The patient was successfully operated on, and a large endothelioma removed from the right hemisphere. It lay immediately underneath the posterior end of the right superior frontal gyrus, extending backward under the upper third of the precentral gyrus and forward into the centrum semi-ovale. It bulged on the mesial aspect of the hemisphere and impinged on the right side and upper aspect of the anterior half of the corpus callosum. After the operation the tonic innervation completely disappeared and a typical hemiplegia of moderate degree, with characteristic reflexes, remained.

In Case 2, a male patient, aged 47, developed a characteristic but not severe Jacksonian attack of the left limbs, beginning in the foot and passing rapidly up to the shoulder, then down the arm to the hand. Gradually progressing weakness of the left limbs followed, the leg being more affected than the arm. At this time, on examination, there was found to be slight double optic neuritis, slight spastic paresis of the left limbs, including the face, and slight relative hypæsthesia on the left side. The tendon reflexes on the left were increased, and an extensor response was obtained. Two months later the weakness on the left side had increased, and the left arm assumed the contraction-attitude of an ordinary hemiplegia. There was, nevertheless, no great loss of strength in it. It was now observed that the phenomenon of tonic innervation was strikingly present in the left upper limb, both in flexor and extensor groups. When the patient put his left hand on his head he was unable to bring the arm down for three minutes, owing to tonic innervation of the muscular groups concerned. The right arm was entirely normal. There was no apraxia or dyspraxia in the left hand, but the tonic innervation interfered with his passing readily from one movement to another. There were no "pseudo-spontaneous" movements, and no ataxia. Fair attempts at writing were made with the left hand. The patient was successfully operated on, and a large endothelioma was removed from the right frontal lobe. It pressed deeply into, but did not invade the cortex. It grew from the falx, and lay exactly over the posterior ends of the right superior and middle frontal gyri, extending to the anterior margin of the fissure of Rolando.

After the operation the phenomenon of tonic innervation entirely disappeared, and a typical slight spastic hemiplegia remained, with characteristic reflexes. Eventually a good recovery was made.

In Case 3, a female patient, aged 58, had suffered for two years from indefinite attacks of cramp in the left thigh and from paræsthesiæ in the left hand. Four months before admission to hospital she had an attack, without loss of consciousness, in which the left arm and leg became powerless, and all feeling left them. Improvement set in, however. It was thereafter noted by the patient that she was unable to relax her grasp easily on the left side, and unable to relax other movements of these limbs. On admission she was found to be rather emotional and inattentive. There was no trace either of motor or of sensory aphasia. She wrote quite well with the right hand. On the left side there was a spastic hemiparesis, the leg being much more involved than the arm, in which considerable power and freedom of movement were still present. The chief motor phenomena were as follows—all confined to the left limbs (arm):—

(1) Severe motor apraxia; complete inability to perform movements to order which she performed automatically without any difficulty; no ideational apraxia, and no ataxia.

(2) Constant "pseudo-spontaneous" and "amorphous" movements.

(3) Inability to imitate movements.

(4) Tonic innervation.

(5) Agraphia.

Apart from the tonic innervation there was some spasticity of the left arm. The reflexes were brisker on the left side than the right, and on the former an extensor response was always present. The condition of sensibility was a little difficult to determine; apparently there was diminution of cutaneous sensibility in the left limbs; there seemed to be impairment of the muscular sense. Stereognosis also appeared to vary. The patient eventually left the hospital practically *in statu quo*.

Considerations suggested by the Analysis of our personally observed Cases.

Before going on to a detailed investigation of the phenomenon of tonic innervation we may here indicate several important considerations suggested by the examination of these cases, which will aid subsequently in coming to our conclusions as to the significance of the symptom.

(1) In the first place, it is clear that tonic innervation may occur by itself, i.e., without either dyspraxia or apraxia. Our first two cases exemplify this well. The two conditions are not causally connected; one is not the inevitable accompaniment of the other. Where the two occur together, therefore, it is possible to separate the mechanisms clinically.

(2) Our first two cases serve to determine the localizing value of the phenomenon; in each case the tumour lay in practically the same position. It will be readily understood that the combination of tonic innervation and of motor apraxia is not unexpected, inasmuch as the site of the lesion in the uncomplicated cases is in the immediate neighbourhood of one of the sites whence motor apraxia may be derived, and our third case shows this.

(3) In all our cases tonic innervation was present in association with slight but unmistakeable involvement of the cortico-spinal path; in each there was slight spasticity of the affected limbs, and characteristic alterations in the cutaneous and deep reflexes were present. At the same time, it should be noted that the greater the involvement of cortico-spinal fibres, the less marked was the tonic innervation. In each case the leg was more severely affected than the arm and the phenomenon was there less in evidence. Where the paralysis was profound the phenomenon was absent. After the operations in the first two cases, the tonic innervation disappeared with the development of a post-operative hemiplegia.

(4) The symptom was evident solely and exclusively during voluntary innervation of the muscular groups concerned. In Case 3, for instance, the patient might automatically put up her hand to rub the point of her nose and do so with the greatest readiness, but the effort to do so with full voluntary intent always resulted in a persistent tonic innervation which effectually inhibited the attainment of the desired end.

(5) The symptom is not necessarily accompanied with, still less derived from, any constant sensory changes in the affected limb or limbs.

(6) In all our cases it was strictly unilateral, and is, therefore, localizable.

(7) We were not able to satisfy ourselves that either electricity or massage had any constant effect in curtailing the duration of the tonic innervation. It may be remarked, also, that the electrical changes described as the myotonic or neuromyotonic reaction were not present in our cases in characteristic form.

Review of Analogous Cases from the Literature.

Hitherto, as far as we are aware, no article has appeared expressly devoted to a consideration of the symptom of tonic innervation, hence it has been a matter of some difficulty to collect cases in which reference to it has been made, and it may be that others, of equal importance, have escaped our notice. We have been able, however, to collate not a few instances of the occurrence of the symptom in association with intracranial disease, and these will now be enumerated.

(1) Liepmann's [26] patient was frequently unable to relax a given innervation of the left upper extremity, which, be it remembered, was not apraxic, or showed only slight dyspraxia. His wife noticed that when he grasped an object in the left hand he was often unable to let it go from his hand, but held it in a tonic cramp. The symptom, however, was apparently neither constant nor notably developed, and the author's references to it are few and not prominent. A complete left hemiplegia, which occurred not long before the patient's death, complicated matters and made further investigation impossible. At the time when the tonic innervation was observed there was slight weakness of the left half of the face and to a less extent of the arm.

(2) Steinert's [39] case (his Case 2) was that of a man, aged 36, with typical signs of intracranial tumour. There was gradual onset of weakness of the right limbs, the leg being more affected than the arm, with slight spasticity of the same. When at rest, they assumed the attitude of hemiplegia to some extent. No sensory changes were noted; the right abdominal reflex was absent and a right extensor response was present. The patient held fast anything he grasped with the right hand, unable to loosen his hold. He had great difficulty in relaxing a handshake. Only with great effort could the examiner release his fingers from the grip of the patient's right hand. There was no apraxia or dyspraxia in either hand, apparently; at least it is not specifically referred to.

The lesion was a glioma involving the mesial aspect of the left hemisphere, destroying the left gyrus fornicatus and invading the corpus callosum from genu almost to splenium; it compressed the gyrus calloso-marginalis, the paracentral lobule, and the internal capsule, and it bulged across the middle line, towards the corresponding gyri of the mesial aspect of the right hemisphere.

(3) In Steinert's experience the symptom is rare; he had only seen it in one other case, to which brief allusion is made. The patient was a male hemiplegic, who was able to extend his paretic arm at the

elbow; his flexors were weak, but even passive flexion at the elbow was thereafter for a short time impossible owing to tonic innervation of the triceps, and that in spite of every endeavour of the patient to relax. The condition gradually disappeared as the patient improved.

Reference is made to it here, although the details are rather meagre.

(4) Van Vleuten's [41] case is mentioned in Wilson's paper on apraxia referred to above. The patient was a man, aged 55, whose symptoms began with difficulty in finding words—slight amnesic aphasia and verbal paraphasia. There was some weakness of the right side of the face, while definite symptoms of involvement of the right limbs, in the shape of weakness or alteration in the reflexes, were at this stage absent. On the right side, nevertheless, two symptoms occurred; viz., moderate tremor of the right arm when at rest, increasing with muscular exertion, and characteristic tonic innervation of the same. If the patient took an object in his right hand he was unable to let it go, in spite of every endeavour and the urging of the examiner; other muscular groups showed the same tonic innervation, and it was present also in the right leg, to a less extent. The tonic innervation mechanically interfered with the performance of movements of the right limb, but there was no apraxia. On the other hand, however, definite motor apraxia was noted in the left arm and hand. The condition steadily progressed, till eventually the patient sank into a state of apathy and stupor. By this time a right hemiplegia had developed and the tonic innervation disappeared: on the left side the apraxia was more complete.

The lesion was a tumour of the left frontal centrum semi-ovale, destroying the white matter underneath the left cingulum and limbic lobe, as well as that of the superior frontal gyrus; it invaded the left side of the corpus callosum from the genu backwards almost to the splenium, and also grew to some extent into the left lateral ventricle. The basal ganglia and internal capsule and gyri centrales were intact, but pressed on somewhat and distorted a little by the pressure.

(5) Kleist [20] has published a very full record of a case on which he bases his conception of "cortical or innervation-apraxia." The patient was a man, aged 44, with a syphilitic history. He developed a left hemiplegia, then later an incomplete right hemiplegia, with difficulty in speaking. On examination there was found to be a moderately severe degree of motor aphasia, with bilateral spasticity of the limbs, increased deep reflexes, and bilateral ankle-clonus. From their hypertonicity the limbs offered considerable resistance to passive movements,

the rigidity resembling that of paralysis agitans, i.e., involving both flexor and extensor groups. The disturbances of motility exhibited by the patient were of great interest, consisting in a form of motor apraxia more pronounced on the right side than on the left. It frequently happened that in attempting to carry out a given movement the patient innervated the wrong group of muscles, and made other mistakes referable to the complexity of the innervation required, which do not concern us further here. In addition, the phenomenon of tonic innervation was present in a well-marked form. When it was desired either to end a given innervation, or to replace it by another, the patient was unable to do so. In dealing cards, for instance, he had the utmost difficulty in opening his closed fingers. In his use of a hammer, the phenomenon was particularly well exemplified. Instead of bringing the hammer down smartly on to the object, tonic innervation and extremely slow relaxation of the muscles that lift and extend the arm resulted in the hammer descending with amusing gentleness. Tonic innervation also occurred in writing.

The symptoms in this case were asymmetrically bilateral. On the left side the slowness of movement, tonic innervation, and difficulty in executing fine movements were more marked, while substitution-movements were better seen on the right. The apraxia of the left upper extremity, in other words, closely resembled what occurs in the limb-kinetic type, and was analogous to cortical hemiplegias or monoplegias with loss of fine movements, while the right-sided apraxia came rather into the category of transcortical motor apraxia (of Heilbronner: Liepmann's ideokinetic apraxia, or motor apraxia *par excellence*). The case was purely clinical, but Kleist postulated lesions more or less resembling those found in Liepmann's case; he thought the left-sided dyspraxia could be explained by a lesion in the left hemisphere; in addition, there must have been a lesion "in or in the immediate neighbourhood of the right central gyri or their underlying white matter." No attempt is made to correlate any of the postulated lesions with the symptom of tonic innervation.

Another case of a differing type, recorded by the same author, will be mentioned later.

(6) A good case has been put on record by Kurt Goldstein [12] [13]. To it our Case 3 bears an altogether extraordinary resemblance.

The patient was a woman, aged 57, who had a stroke on the left side. The left leg remained badly paralysed, while the condition of the face and arm cleared up to a large extent. The following phenomena, however, remained, and concerned the left side only:—

(*a*) Severe motor apraxia; there was great difficulty in the performance of movements voluntarily which were accomplished automatically with ease.

(*b*) Frequent "pseudo-spontaneous" and "amorphous" movements.

(*c*) Poverty of spontaneous movements and marked impairment of "movement-intention." When the patient made any voluntary movement correctly to order, she regarded her success as accidental. There was no "will-feeling" in her spontaneous acts.

(*d*) Inability to imitate movements.

(*e*) Tonic innervation in many movements of the left side.

(*f*) Agraphia.

There was slight cutaneous hypæsthesia on the left side, with considerable diminution of pressure-pain, as well as of the sense of movement and of passive position. Localization, too, was defective. The tendon reflexes were increased on the left, and the plantar response was doubtful. Her left hand was constantly catching hold of the bedclothes in pseudo-spontaneous fashion. When objects were grasped in her left hand she was unable to let them go. Once she thus caught hold of her neck and "nearly strangled herself." She opened her mouth readily when eating, but could scarcely do it on request, and so on. There was no ataxia. The patient was "incontinent" of urine and fæces, but this was probably apraxic in origin; she would sit on the commode for an hour without any result, then was "incontinent" as soon as she was put back to bed.

In almost all its details this case shows a remarkable similarity to our third case, hence the value of the pathological findings is enhanced.

The lesion was a large softening in the distribution of the posterior branch of the right anterior cerebral artery—the branch to the corpus callosum. The softening extended along the mesial aspect of the right hemisphere, destroying the gyrus fornicatus and its white matter, the posterior extremity of the superior frontal gyrus, part of the cortex and white matter of the paracentral lobule, as well as the right hemisphere part of the corpus callosum in almost its whole extent. The central gyri, otherwise, and the internal capsule and basal ganglia, were untouched.

(7) In Kroll's [24] paper there is a case (his Case 3) which has some bearing on our subject. The patient was a man, aged 45, in whom gradual weakness, first of the right side and then of the left, developed. On examination there was slight motor and sensory aphasia,

slight right hemiplegia, with increased tendon reflexes and moderate spasticity. Sensibility was normal. The patient was absolutely blind. There was very definite motor apraxia of the right limbs, and also to some extent of the left. Tests for bilateral movements brought out the apraxia very well. He made all sorts of "pseudo-spontaneous" movements with the left hand, but was incapable of accomplishing any correctly on request. Imitation of movements passively impressed on either side was impossible. According to Kroll the patient also showed constant evidence of Liepmann's "tonic perseveration," apparently with both right and left limbs, but it is not certain that the author is describing the phenomenon with which we are here concerned. Thus the patient frequently held a limb, after moving it, in a given position for a considerable time, but it is not clear whether he simultaneously endeavoured voluntarily to relax the innervation and failed to do so. "In all his movements the patient evinced a tendency to maintain any given pose of his limbs once it was assumed, e.g., he held his fingers for a long time spread out." Kroll calls this "tonic perseveration," but in the absence of details as to the muscular condition, one may take leave to be dubious, the more so as it is stated that "the at first strongly marked apraxia gradually changed into a practically constant state of tonic perseveration." The condition, in our opinion, appears rather to have been analogous to the "Willenlosigkeit" of some of Pick's patients (see below), where movement came to a standstill altogether.

The lesions were softenings in the left middle frontal gyrus, in the left supramarginal gyrus, and in both right and left angular gyri, involving the optic radiations. In addition, the whole of the right hemisphere part of the corpus callosum was disintegrated.

(8) A similar uncertainty exists in regard to a case reported by Coriat [7] (his Case 1). The patient was a young man, aged 24, with symptoms of intracranial tumour. There had been transient paralysis of the left limbs, in which there was considerable defect of cutaneous sensibility. On examination of the patient's motility it was found that typical motor apraxia of the left arm and hand existed, while the right side was normal. There was no ataxia. "In spite of the . . . slight weakness and spasticity of the left arm, he could spontaneously lift it above his head, flex, extend, pronate, supinate, fairly well extend and flex the fingers and give a fair grasp, although there was a disinclination on the part of the subject to use this left arm spontaneously." It is doubtful whether the author is employing the term under discussion

in the sense accepted by German authorities and by us when he goes on to say, "This tendency to akinesis was probably due partly to the patient's appreciation of his localized motor apraxia, and partly to a state of tonic perseveration." And to exemplify the latter, he adds: "The left-sided apraxia became more marked when the patient's eyes were closed. . . . A key, a match, and a drinking glass were used correctly with the right hand and decidedly awkwardly and fumblingly with the left. When attempts were made to use these same objects with the eyes closed the left hand seemed to become petrified, as it were, after an abortive start. In other words we seem to have here the phenomenon of tonic perseveration." "Sometimes the subject becomes petrified, as it were, in the attitude of executing a simple or complex act either requested or spontaneous; this is known as tonic perseveration."

We are of the opinion that in this instance also the condition is that where voluntary action comes to a standstill, a condition to be sharply differentiated from tonic innervation.

(9) Rhein's [35] case was unfortunately even more than usually complicated; nevertheless, his patient exhibited true tonic innervation in the left arm and hand.

The case was that of a man, aged 55, who when he came under observation was quite blind; he had complete loss of the sense of position, could not localize any cutaneous stimulus, and did not appreciate touch or painful stimuli in the left arm. There was also complete astereognosis. There was no paralysis, but the left arm was rarely if ever moved voluntarily. The right arm was apraxic; with the left he was frequently seen to make automatic or reflex movements correctly enough. "When asked to grasp the hand of the examiner with his left hand he occasionally succeeded, and at such times he would not relax his hold, apparently involuntarily in fact, grasping more and more tightly as long as the hand of the examiner remained within his grasp. This was undoubtedly a manifestation of perseveration, of the tonic variety described by Liepmann." No ataxia was observed. The left limbs were rather more spastic than the right; the deep reflexes were increased, and there was no extensor response.

"The autopsy revealed the presence of degeneration of the white matter of the right occipital and parietal regions, on the convexity, and the posterior portion of the temporal lobe. . . . On the left side there was degeneration in the occipital and temporal regions to a much less degree. . . . The corpus callosum, in its posterior portion, was degenerated. . . ."

Rhein quotes Liepmann's view that tonic perseveration is due to disturbance of the motorium itself, and considers this opinion confirmed "somewhat" by the pathological findings in his case, in which the right postcentral gyrus was in part degenerated: "that is, provided we believe that the motor centres are not entirely confined to the precentral region." But Rhein himself also states, in his description of the autopsy, that "the inner surface of the dura was covered with a thick plastic exudate, *extending over the entire surface of the brain on the right side*, an intense internal pachymeningitis" (italics ours). Evidently this pathological condition ought not to be ignored in a consideration of the clinical import of the lesions. In any case, the profound sensory changes in the left arm (as elsewhere) complicate the purity of the symptom.

(10) An interesting case of agraphia from frontal tumour has been published by Macfie Campbell [6]. The patient was a woman aged 55, with general symptoms of increased intracranial pressure. She showed a writing defect which was not explained by the presence of either a motor or a sensory aphasia. There was no apraxia. Dysarthria was present, and there was some weakness of the right side of the face, and of the right limbs. It was noticed that there was a tendency to (true) perseveration, for instance in answers to questions; and in addition "the motor condition of the right hand was interesting; when the strength was tested at first slight weakness was observed, but in a little while the grip seemed nearly as strong as that of the left hand; when told to let go, the patient did not at once relax, but required to be urged to let go." And again: "After admission to the hospital there was noticed the same tendency to motor perseveration and also *inability to relax promptly* as well as a want of spontaneous utilization of the arm, without there being any reduction in the muscular force of the arm when innervated" (italics our own).

Although the details would have been more valuable to us had they been more specifically dealt with, it is possible to claim this case as one of tonic innervation of certain groups in the right arm.

The lesion was a large cyst of the left frontal lobe, separating the superior and middle frontal gyri; its posterior limit corresponded with the central fissure. The mesial aspect of the left hemisphere bulged inwards and downwards, and there was a corresponding depression of the opposite hemisphere.

(11) In Pick's [33] "Studien über motorische Apraxie" some cases are described which have a bearing on the problem before us.

One of his patients (case Prokop), asked to lie down on his bed, lay down with his right leg stretched in an awkward position over the edge; it was held so, immobile, for more than two minutes. The same patient, asked to take a drink out of a jug on the table, went and put his lips and face into the mouth of the jug and remained so practically without moving. Another patient (case Havranek), asked to light a candle, held the match alight in her right hand, in the neighbourhood of wick, quite immobile, until the match actually burned her fingers.

These incidents are quoted because they exemplify persistence of a given innervation-complex, but at the same time they must be differentiated from the phenomenon of tonic innervation, to which the resemblance is but superficial. The persistence of an attitude, the akinesis following on movement, is not maintained *in spite of* the patient's every endeavour to inhibit the innervation, but from a defect on a higher plane, a "will-lessness" which is not identical with inability to execute the behests of a normal "will" as in tonic innervation.

(12) A case which has been published by Kleist [21] under the title of "Nachdauernde Muskelkontraktionen" may finally be alluded to.

The patient was a woman, aged 29, who from about the age of 16 had suffered from a certain awkwardness of hand movements, and noticed she could not relax her grasp properly. In addition there were weakness and undue fatigability of many of her muscles. On voluntary contraction of the small muscles of the hands, flexors and extensors of fingers, biceps, triceps, pterygoids, peroneus longus and brevis, &c., they passed into a condition of myotonia. As a rule, the more successive identical movements were made, the worse the myotonus; in this respect, therefore, the case differed from the classical myotonia congenita. Certain muscle groups, including the sternomastoids, the dorsiflexors of the feet and the extensors of the arms and the abdominal muscles were atrophic. In addition, kypho-scoliosis, pes equinus and claw-foot, slight peripheral contractures in the arms, paresis of the lower face, slight bilateral ptosis, slight sensory changes and vasomotor disturbances were noted.

The exact nature of the case is not clear. The author considers it an atypical myotonia: Batten and Gibb [1] include it, under reserve, as a case of myotonia atrophica, while Bramwell and Addis [3] place it definitely in their group A, comprising forty-five recorded cases of myotonia atrophica resembling each other more or less closely in the matter of the distribution of the atrophy.

TABLE OF REPORTED CASES OF TONIC INNERVATION.

No.	Author	M. or F.	Side affected	Pyramidal affection	Apraxia	Nature of lesion	Site of lesion
1	Goldstein...	F.	Left arm	Slight weakness of left arm, much more of leg	Left motor apraxia	Vascular	Softening of right superior frontal gyrus, gyrus fornicatus, white matter of paracentral lobule, and whole of right side of corpus callosum.
2	Kleist ...	M.	Left limbs chiefly	Some left hemiplegia, also right	Left limb-kinetic apraxia, right motor apraxia	Syphilitic	(Clinical case.)
3	Liepmann	M.	Left arm (occasional)	Slight weakness left face and arm	Gross apraxia right, slight dys-praxia left	Vascular	Widespread softenings: left frontal and parietal, corpus callosum, right internal capsule.
4	Rhein ..	M.	Left arm	Slight spasticity of left limbs	Right apraxia, left (?) (case complicated by gross sensory loss)	Pachymeningitis	"Over entire surface of brain on right side," &c.
5	Steinert ...	M.	Right arm	Progressive right hemiplegia	?	Tumour	Left mesial gyri anteriorly, corpus callosum: left paracentral lobule and internal capsule pressed on.
6	van Vleuten	M.	Right arm, slight in right leg	,, ,,	No apraxia right, definite motor apraxia left	,,	Left frontal semiovale, white matter under superior frontal and mesial gyri, left side of corpus callosum; left internal capsule pressed on.
7	Wilson and Walshe	M.	Left arm	Slight affection of left arm, much more of leg	None	,,	Posterior end right superior frontal gyrus, under mesial gyri, at corresponding level, pressing on upper precentral gyrus and right corpus callosum.
8	,, ,,	M.	,,	Slight spastic paresis left arm, more of leg	None	,,	Pressing into posterior ends of right upper and middle frontal gyri, extending to anterior margin of fissure of Rolando.
9	,, ,,	F.	,,	Slight spastic paresis of left arm, much more of left leg	No apraxia right, severe motor apraxia left	Vascular	(Clinical case.)

Now, the interest of the case consists in the association of disease of the central nervous system and myotonia, and the author is at pains to argue that the latter is of central origin and due to impairment of function of the fronto-pontine-cerebellar system. To this matter discussion is directed in a subsequent section.

The cases of tonic innervation here collected are evidently closely related; in each the phenomenon was strictly unilateral, and it was accompanied by other signs pointing unequivocally to the existence of a central lesion. Leaving out those which are not quite sufficiently definite for our purpose, or sufficiently detailed, we have a record of nine—Liepmann 1, Steinert 1, van Vleuten 1, Kleist 1, Goldstein 1, Rhein 1, and Wilson and Walshe 3—on which to base a study of the symptom. And further, of these 5 came to autopsy, while 2 were successfully operated on; hence we have no fewer than 7 to aid us in a consideration of the localization of the phenomenon.

These nine cases belong admittedly to one type, clinically speaking; the occurrence of tonic innervation, or something very like it, in certain other diseases of the central nervous system, will occupy attention in a subsequent paragraph.

Terminology and Relation to Perseveration, properly so-called.

The term "perseveration" ("perseveratorische Reaktion") was apparently first used by Neisser [30] to express that condition in which the patient repeats a particular act when requested to perform another act, or when he spontaneously wishes to perform another act. To quote from Wilson's [42] paper on apraxia: "The persistence of certain impressions may become a fertile source of error in the execution of complex movements; a patient may recognize an object and use it correctly, but be unable to prevent himself from repeating this act when a different object is presented to him. He may use a spoon properly, but repeat the action of feeding himself with the next thing he is shown, which may, for instance, be a corkscrew." Whether the persisting idea arises spontaneously or secondarily to sense impressions is indifferent. Numerous instances of perseveration are given in the paper from which this excerpt is taken, and to it the reader is referred.

To this variety of perseveration Liepmann [27] has given the name of "intentional perseveration," because it is characterized by the reproduction of a given movement-complex or act when, and only when,

another is intended. The repetition is not independent or spontaneous, so to speak; it occurs only when some new act is intended, and in place of that act, otherwise it would not be noted. It is the commonest form of perseveration, and is well known to the student of nervous and mental disease. According to Liepmann it is found associated mostly with lesions in the posterior third of the brain, especially in the temporal and parietal regions. In our experience it is most often an accompaniment to a degree of agnosia of the auditory or visual variety, but we have also observed it in certain diffuse or generalized intracranial conditions, and we are of the opinion that in the present state of our knowledge it cannot be even tentatively localized.

The term "intentional perseveration," in English at least, is not a satisfactory expression, as naturally it might be taken to signify that the perseveration is intentional, whereas the exact contrary is the case. It is more in accordance with the facts, and at the same time non-committal, to describe this variety simply as "perseveration" without an attached epithet, and this we propose to do.

A second form of perseveration is that in which the patient continues making a particular movement or movement-complex though told to stop: Liepmann's patient, for instance, continued writing hooks and letters though requested to cease writing. The patient cannot put the volitional brake, as it were, on a movement-complex once initiated, but continues to repeat it without apparently making any effort to check it. It is not, however, necessary that the element of continuance against request should enter into the phenomenon. Campbell's patient "continued to shake hands for an unusual length of time." A patient observed by Wilson, writing her name "Winnie" wrote "Winninninninn . . " indefinitely. A patient of Breukink's [5], peeling potatoes, went on peeling indefinitely, apparently unable to cease. This "Iterativerscheinung" has frequently been noted in cerebral and mental disease.

These varieties of perseveration may occur, as the reader will have seen, in other spheres than that merely of limb-movement. In writing and in speaking, they may also be observed. As the field of speech, however, with its "verbigeration," its "recurring utterances," and its "word intoxication," is somewhat more complicated, it may be left to one side for the present.

Liepmann has proposed to denote the second variety "clonic preservation," because there is alternation or cessation of innervation and then again reproduction of the same. The "tendency to innerva-

tion" persists; the alternation of contraction and relaxation constitutes the "clonic" element in the phenomenon. He opposes it to "tonic perseveration," which, as we have stated, we much prefer to designate "tonic innervation."

We are of the opinion that the expression "clonic perseveration" is as likely to lead to misunderstanding as "intentional perseveration," though for a different reason. It is clear that so-called "clonic perseveration" is a phenomenon of a different nature, on a different plane, from "tonic perseveration," or better, tonic innervation. "Clonic perseveration" is very closely allied to perseveration *sensu strictiori*, in fact, scarcely to be distinguished from it, in certain cases at least. In any case, the terms "clonic" and "clonus" should be reserved for phenomena occurring in the lower levels of the nervous system, and they are erroneously applied to phenomena which are essentially of a psychical or psychomotor nature.

A consideration of the nature of perseveration justifies our assertion that these two varieties are much more closely related to each other than either is to tonic innervation. They are both phenomena which are dependent on a disturbance on the psychical level. They result from the persistence of an idea: Liepmann says it is the "tendency to perseveration" that persists. There is fixation of idea, and as a result the patient executes in succession a series of more or less identical movements whatever be the stimulus on the afferent side. The defect is in the intra-psychical limb of Wernicke's reflex arc. Whether the patient keeps on shaking hands, or shakes hands each time he is asked to perform some other act, the impairment of function derives from a disturbance of psychical order.

Opinions differ as to the explanation of this psychical defect. It is well recognized that an idea possesses a certain duration beyond the time of its clear presentation to consciousness, and Liepmann suggests that the idea of a given movement-complex appears to persist abnormally, and is translated into action again and again, because there are hindrances to the carrying out of other innervations along other paths. As one of his patients made the same perseveration movement first on one side and then on the other, it is clear that not the limb-kinetic element in the movement, but the idea of the movement itself, was at fault, otherwise the phenomenon would have been unilateral. In another place Liepmann postulates an "Unwegsamkeit vieler Bahnen" to explain the persistence of a particular act. Von Sölder [38] is of the opinion that perseveration has its origin in the *active* preponderance

of the persevering idea. In a not dissimilar way Douse, quoted by Pick [33], explains what he calls "opisthomimesis," a condition identical with perseveration. "The mind lags behind the hand and perturbs its action by surviving impressions of what has been already written." Pick [32], on the contrary, holds the view that there is *passive* preponderance of the persevering idea. As a consequence of the diminution of intensity of other ideas, from fatigue, the persevering idea is relatively reinforced and may remain some time. It should perhaps be mentioned that in explaining this diminution of intensity of other ideas as arising from fatigue Pick is arguing from the circumstances surrounding the appearance of perseveration in post-epileptic confusional states, in which he has often found perseveration to occur. Elsewhere, however, he emphasizes the importance of attention in this connexion, and adduces evidence showing that defect of attention may be a factor in perseveration. The patient may be, as it were, in a state of distraction; associations that ought normally to be aroused by the presentation of subsequent ideas are pathologically feeble, and perseveration results. Stoddart [40] speaks of "ideational inertia" as tantamount to perseveration, and his use of the phrase shows that he regards it as equivalent to ideational persistence or persistence of an idea. Some confusion, perhaps, may arise in connexion with the word "inertia." It is a little difficult to understand why inertia of an idea should occasion the actual repetition of the movement-complex which depends on it. An apposite citation from James [18] is given by Pick: "the fact that a certain intensity of the consciousness is required for its impulsiveness to be effective in a complete degree. There is an inertia in motor processes as in all other things. In certain individuals and at certain times (disease, fatigue) the inertia is unusually great, and we may then have ideas of action which produce *no* visible act" (italics ours).

This leads us to a last point in the matter of perseveration. In the cases of Kroll, Coriat, and Pick, quoted above, reference has been made to an interesting symptom characterized by apparent cessation of action; during or at the close of the performance of a given act the patient comes to a standstill—either a limb or limbs, or the whole person, as the case may be—in a particular attitude, which is maintained almost indefinitely. The incidents quoted above as illustrative of this condition need not again be particularized. Pick [33], who has devoted much attention to this phenomenon, regards it as a perseverational reaction, and considers that several factors combine to produce it, or rather, that in different cases different factors are at work. He is not certain

whether to consider this "Untätigkeit" the outcome of will-lessness ("Willenlosigkeit") or not, yet he inclines to emphasize the analogy between the state in question and the absence of spontaneity or initiative seen in other cases, in which there is doubtless a true volitional defect.

It would lead us rather away from the main object of this paper to discuss the question of absence of initiative or spontaneity, with its resultant akinesis, in relation to perseveration. Akinesis has frequently been noted in intracranial disease (Hartmann [16], Wilson [42], and others) more especially in connexion with lesions of the frontal lobes and corpus callosum. Etymologically speaking, where action is in abeyance altogether, or where it comes to a standstill prematurely, we are no doubt dealing with a condition of apraxis, and so akinesis may be regarded as true apraxia. But as it is so often associated with a state of generalized mental hebetude and deterioration, no good purpose can be served by an endeavour to link it to apraxia in all cases. Complicating factors, such as inattention and incapacity for retaining impressions, intervene to render its analysis a matter of peculiar difficulty. We are of the opinion, further, that this holds good for that condition mentioned above, which is not, we think, entirely identical with akinesis; the instances referred to above exemplify a condition in which there is normal initiative to begin with, but it speedily exhausts itself; further, sometimes the required movement has been actually completed before the condition of akinesis supervenes.

It is sufficient, then, for our purpose merely to allude to these matters and to point out that whether the cessation of action in these cases, and the passive maintenance of an attitude, is a form of apraxia or akinesis, or not, the phenomenon is at any rate essentially one of a psychical order. Objections might be raised to the view of Pick that it depends on momentary "will-lessness." Though movement come to a standstill visibly, none the less continuance of innervation may be producing this. Liepmann questions whether an awkward attitude would be so preserved in the absence of impulses from the cortex. It may, however, be automatically preserved, no volitional element entering into it, just as a movement may be automatically performed, which the patient is unable to execute on request.

Allusion has already been made to Pick's description of this "akinesis" as a perseverational reaction, which adds yet another connotation to the term perseveration. We are much inclined to

suggest that varieties (1) and (2), viz., the repetition of a given movement in place of another, and the continued repetition of a given movement when in a normal individual it would cease, should be known as *active perseveration*, whereas variety (3)—viz., the cessation of action which results in the maintenance of an attitude either in the middle or at the end of a given movement-complex—should be known as *passive perseveration*.

The description which has been furnished of these phenomena will, we think, have made it obvious to the reader that they are essentially of a psychical nature, and the fact that they are the outcome of a psychical defect distinguishes them *in toto* from tonic innervation—Liepmann's tonic perseveration—where the innervation continues though the patient voluntarily makes every endeavour to inhibit it.

Nature of the Phenomenon and Relation to Apraxia.

In our first and second cases the phenomenon of tonic innervation occurred in a pure form. They were uncomplicated by any disturbance of a psychical order, volition in the psychical sense was intact, and no impairment of attention or memory or capacity for retaining impressions was noted. With the best will in the world and with full insight the patients were still incapable of immediately relaxing or inhibiting the contraction of a given muscle or group of muscles on the affected side, although the antagonists were being strongly innervated. The symptom, however, was not always present in unvarying intensity; some attempts at movement in the affected groups were better than others, while occasionally the patients were seen to move the limbs with comparative ease, but this never occurred during examination. In our third case the symptom was observable in equal degree, and, though complicated by other symptoms, its value as compared with the first two is enhanced in that it demonstrated in unmistakeable fashion that the phenomenon was confined to volitional as opposed to automatic or involuntary movements. The patient was incapable of opening her voluntarily closed fist, but on the request "Look at your hand," delivered smartly and unexpectedly, she would automatically open the hand and look at it. She would hold on to the bedclothes interminably, until in response to some stimulus relaxation unexpectedly occurred and the hand was brought up to her face to rub the point of her nose. Similarly, in the first two of our cases, when the limb was left to itself a tonic innervation soon, sometimes immediately, relaxed altogether, in a quite involuntary way.

In a word, the volitional element is the disturbing element; it has been frequently remarked, both in our own cases and in others, that as the patient tried to relax so his grasp perceptibly tightened: and we think the conclusion is justifiable that the variations in duration and intensity are largely due to variation in volition and in voluntary attention.

In saying this, however, we wish it to be clearly understood that we do not mean to imply that imperfection of volition is the *cause* of the phenomenon; if a patient knows what he wishes to do, and voluntarily (and correctly) innervates the antagonists in an attempt to overcome the tonic innervation, his volition is unimpaired; we mean simply that the condition varies with volition, but is not directly dependent on it. In the same way an individual may know a piece of music "by heart" and play it through automatically without being able to begin voluntarily in the middle of it. It is well known that the direction of voluntary attention to an act, in the case of a patient with motor apraxia, may result in that act becoming the more confused. In one of Bonhoeffer's [2] cases the more the patient tried to imitate movements made in front of him the worse was the result. Liepmann's patient was able to execute acts "spontaneously" which he quite failed to accomplish on request. Pick quotes the following passage from a paper by Bernard (which we have not been able to obtain); it is in the words of the patient himself: "I understood perfectly what was said to me and my intelligence appeared to me intact. Yet, having in my hand a common household article, the idea of how to use it completely escaped me, but I had sufficient presence of mind to remember that the best way to get the idea back would be to use the article mechanically without paying the least attention to it: the result was quite successful."

The symptom of tonic innervation, then, is aggravated by voluntary attention, just as other symptoms may be, but volition itself is intact.

The fundamental defect in tonic innervation is not of a psychic, but of a psychomotor nature; the patient is unable to inhibit an innervation which he has correctly initiated. As the affected groups remain in a state of active contraction, there must be continuance of stimulation or innervation at the source of movement, but that this cannot represent a permanent defect in the middle level centres—the motor centres—is plain when we remember that with automatic movements the phenomenon is not observed. We are dealing, therefore, with a psychomotor as opposed to a motor condition, one which corresponds, physiologically

speaking, to an impairment of function at the distal end of the psychomotor (efferent) limb of Wernicke's reflex arc.

We have seen that tonic innervation may occur independently of either dyspraxia or apraxia. Our first two cases prove this definitely. Any awkwardness of movement of the left limbs in these cases could be accounted for by the great difficulty experienced by the patients in passing from one movement to another; the attempts they made to execute given movements were always very fairly recognizable. Similarly, van Vleuten's patient presented the symptom in his right limbs, which were eupraxic, while his left limbs were apraxic, but there tonic innervation was wanting. Again, Liepmann's patient had tonic innervation in the limb which was but slightly dyspraxic, while the right arm was grossly apraxic, without, however, showing tonic innervation.

The symptom, therefore, sometimes is independent of any concomitant apraxia, and it sometimes occurs in cases where apraxia is conspicuous by its absence. On the other hand, in our Case 3, as in Goldstein's case, and in several of the other cases, the patient was undoubtedly apraxic in the same limb or limbs as presented the symptom or condition of tonic innervation. In view of these facts the latter must be considered an independent symptom and should apraxia occur with it the two should be dissociated for clinical purposes.

Relation to the Pyramidal System.

In no one of our three cases was the cortico-spinal system functionally intact on the side which was characterized by the occurrence of tonic innervation on volitional movement. That is to say, an extensor response and ankle-clonus were present, and in the case of the affected arm the tendon reflexes were increased. At the same time, in all three the leg was definitely more involved in the pyramidal defect than the arm, in which little real weakness was to be detected. It is important to bear in mind, also, that as the pyramidal lesion progressed the phenomenon in question diminished, and, in our first two cases, it disappeared after the operation, when a typical hemiplegia developed. From a consideration of our own cases, therefore, the deduction may be drawn that tonic innervation only occurs when the specific functions of the cortico-spinal system are slightly impaired.

This view is borne out by a survey of the other cases in the series. As hemiplegia developed on the right side in van Vleuten's case, so did

the tonic innervation disappear; it was absent when the right hemiplegia was typically present. In this case, it is true, the description given suggests that the specific functions of the pyramidal system may have been normal when the symptom of tonic innervation was first noted. In Rhein's case there was a hemiplegic attitude and some rigidity on the affected side; but the extensor response came later. In Kleist's case the phenomenon was present on both sides, though unequal; the history was one of hemiparesis first on one side and then on the other, and on examination there was slight bilateral spasticity, with brisk jerks and double ankle-clonus. Goldstein's case was one where a left hemiplegia had largely cleared up in the arm and face, while the leg remained badly paralysed. Tonic innervation was present in the arm. Steinert's patient had a right hemiparesis, the leg being more affected than the arm; a right extensor response was present. Liepmann's patient had slight weakness of the face and (?) of the arm at the time when tonic innervation was noted to occur.

The collected evidence, therefore, indicates that the cortico-spinal system for the limb in which tonic innervation is found is not functionally normal; at the same time the involvement must not be too great. Whether the symptom may occur with an intact cortico-spinal system is a little doubtful.

As a corollary, the remark may be made that tonic innervation is not dependent on, nor does it proceed *pari passu* with, hypertonicity of pyramidal origin. The tonus of the affected musculature was only slightly augmented in our cases as in the others that have been recorded. In our Case 3, perhaps, it was rather more marked, as in Kleist's case; yet no definite relation between it and the symptom can be detected. With rigidity and contracture of the right limbs, in van Vleuten's case, there was no tonic innervation on volitional movement.

Evidently no association between hypertonicity and tonic innervation can be established. In progressive lenticular degeneration, as observed by Wilson [43], the musculature is in a state of considerable hypertonicity, but tonic innervation is not found. Whether the latter ever occurs with normal tonus, or with hypotonus, is, again, a little doubtful.

Relation to Myotonia, properly so-called.

One of the most interesting questions in connexion with the phenomenon of tonic innervation is that of its relation to myotonus or myotonia. It was remarked in the introduction to this paper that as

far as the symptom itself is concerned there is little clinical differentiation to be made between it and the myotonus of Thomsen's disease or of myotonia atrophica. The analogies and differences between these we shall now proceed to examine. It should be clearly understood that we are here concerned with a consideration of the symptom only; the complete clinical picture of myotonia congenita or of myotonia atrophica is very different from that of tonic innervation as exemplified by our cases, and no difficulty in reaching a diagnosis should occur.

In myotonia congenita, when the patient suddenly attempts an energetic voluntary movement, the muscles concerned, after being correctly innervated, remain in a state of tonic contraction for a variable period, and although the antagonists are duly innervated the patient cannot relax or inhibit the tonic contraction of the affected group or groups. Such factors as emotion, self-consciousness, cold weather, previous long rest, &c., seem to have an aggravating effect on the symptom, but, speaking generally, the influence of these is neither constant nor very great. The degree to which the volitional element enters into the movements concerned is not important, for, while it is true, as has just been remarked, that the phenomenon comes out with greater distinctness when the movements are forceful, yet it may be seen in more automatic and reflex movements, as in walking, eating, &c. As a rule, also, the impeded movement becomes easier and easier with repetition, until eventually it can be performed in a normal fashion.

In myotonia atrophica, also, the exact relation of which to Thomsen's disease is still in dispute, the myotonic phenomena are practically identical. There is often embarrassment in such imperfectly volitional movements as mastication, moving the tongue, blinking the eyes, &c.

While admitting that the muscular atrophy of myotonia atrophica, the muscular hypertrophy of myotonia congenita, the familial features of these diseases, the peculiarities of the electrical reactions, and so on, will enable the observer to differentiate between them and tonic innervation, we maintain, nevertheless, that as far as the actual symptom of inability to relax a contracted muscular group is concerned, no clear or unequivocal distinction can be drawn between its manifestation in the latter of these conditions as opposed to the former. There is no peculiarity in the symptom, once it occurs, in any one of these diseased states in comparison with the others. The important fact of the occurrence of tonic innervation, generally speaking, only with volitional movement, and not at all with automatic or reflex movement, is an indication that the disturbance of function is cortical or transcortical

in origin, but not that the mechanism involved has a different site from what it possesses in myotonia atrophica and myotonia congenita.

What is the lesion in these two diseases? It has been held very widely that in the latter the essential morbid change is hypertrophy of muscle fibres and that the disease is myopathic. The suggestion has been made, it may be noted, that this hypertrophy of muscle fibres as found at biopsy, is the result of mechanical irritation during excision. Oppenheim and Siemerling [31] have shown that in portions of muscle excised from a living patient an apparent hypertrophy is produced by the contraction of the fibres, which can be avoided if the contraction be allowed to relax. Curschmann [8] has also advanced arguments against the myogenic nature of the affection, and affirms his belief in its central or supranuclear origin. New light has been thrown on the subject from another direction. The investigations of Piper [34] with the string galvanometer have shown that impulses at the rate of some 50 a second reach a voluntarily innervated muscle from the central nervous system. This 50-a-second rhythm is not of myogenic origin. Gregor and Schilder [14] have examined by this method an advanced case of myotonia atrophica, and have shown that during the continued tonic contraction in the affected group the same action-currents of some 50 a second are demonstrable as during prolonged voluntary innervation. In other words, the tonic contraction of myotonia is not a muscular phenomenon pure and simple, but is of central origin. Hirschfeld [17] has also found normal action-currents in cases of myotonia. These physiological investigations are of considerable significance, in our opinion, especially as there is clinical evidence pointing in the same direction. Myotonia has been described several times in association with syringomyelia (Handelsman) [15], in fact a special type has been described as myotonia syringomyelica by Schlesinger [36]. It has been seen in transverse myelitis (Frohmann) [11] and in spinal tumour. As yet apparently only one case of myotonia atrophica has been examined pathologically, by Steinert. In addition to muscular changes associated with the extensive atrophy in the case, there was a well-marked degeneration of the posterior columns of the cord, not unlike what may be seen in tabes. Steinert suggests that this degeneration is not accidental, but possibly an integral feature of the disease. From these facts it may be fairly concluded that there is at least as much evidence to point to a central origin of the myotonic phenomenon as to suggest that it is peripheral.

The view that we take is that the mechanism involved in the

production of myotonia is a spinal cord mechanism, and that the impairment of innervation is an impairment of function of the final common path, to use Sherrington's [37] terminology. Myotonia may occur with spinal cord lesions, and in myotonia congenita there is physiological proof that the defect of innervation is central and not muscular. We are also of the opinion that tonic innervation and myotonia are symptomatologically very closely allied, and that disturbance of the spinal cord mechanism concerned may occur by reason of a lesion at the proximal extremity of the upper motor neurone, as well as by lesions at its distal end. Hence the lesion producing tonic innervation may be cortical. We shall amplify these views in the section that deals with the pathogenesis of the symptom.

Relation to certain Involuntary Movements.

Another interesting question concerns the relation of athetosis, in some cases, to the phenomenon of tonic innervation—a relation which also has bearing on the localization of the symptom.

Some cases of athetosis present a condition identical in every particular to tonic innervation. Thus a patient under the care of Wilson at the National Hospital suffers from right hemiplegia with athetosis, dating from an attack of encephalitis at the age of 8 or 9. The hemiplegia is slight in degree. The patient cannot easily make flexion movements of his right fingers and hand; when he tries, as a rule extension of the wrist and fingers is the result. If, however, he closes the right hand passively with his left, he can then maintain the flexion of the right hand and fingers very well, against resistance. As a rule, further, he can readily open the right hand once it is thus closed. But every now and then he is unable to inhibit the innervation of the flexor groups concerned. The more he endeavours to relax the more strongly does he grasp; if the examiner's fingers are in the patient's hand he can easily convince himself of this. In spite of the tendons of the extensors standing out in contraction, the flexors cannot be relaxed; they may continue for many seconds so that, in fact, the patient says, "My hand remains closed *until I forget about it*, then it opens at once."

An analogous case has been recorded by Kaiser [19], a case of right-sided athetosis and myotonia in a youth, aged 19. "After the muscles are relaxed as completely as possible the patient is asked to execute suddenly any simple movement of the right arm or leg; this is promptly performed, as in any normal individual; thereafter, however, he is

unable to relax the muscles once they are contracted." Grasping a pencil in his right hand, he was unable to let it go for as long as thirty seconds, in spite of immediate innervation of the extensors; "Man sieht wohl, wie die Extensoren sich sofort bretthart anspannen, es bleiben aber die Flexoren ebenso stark contrahirt." The same phenomenon of tonic innervation was present in other muscle groups.

A remarkable case has been published by Mills [29] under the title of "Myotonia and Athetoid Spasm," which is difficult to classify; indeed, it is perhaps unique. The patient suffered from athetoid movements of the left limbs, and involuntary tonic contractions of the muscles of that side effectually prevented him from accomplishing any given voluntary movement satisfactorily. If he wished to grasp an object the extensors would spring the hand open and would remain tonically innervated in spite of every endeavour of the patient to relax or inhibit them. It is not quite clear from the description given by the author whether the phenomenon of tonic innervation, as we have been describing it, existed in this case; it occurred apparently with involuntary as opposed to voluntary innervation, but the whole case is so curious that we are not disposed to lay stress on it. Nevertheless, the indications all pointed to the symptoms being of central origin.

An equally curious case, at one time diagnosed as atypical myotonia, has been recorded by Bremer and Carson [4]. The patient, in addition to suffering from left-sided tonic spasm, sometimes of the sinuous nature of athetoid movements, was frequently unable to relax innervation on both sides of the body. When bringing an axe down on to a block of wood he became immobilized for many seconds in the attitude. The lesion was an angioma of the right cerebral motor area.

A distinction must be drawn, in cases of athetosis, between mere involuntary mobile spasm and the phenomenon under discussion. In athetosis the involuntary innervation of a muscle-group will immobilize the limb, or a segment of the limb, in an athetoid attitude, as in the case of a patient seen by Wilson whose fingers, after a stroke, very frequently assumed a fan-shaped attitude of separation, and it was with difficulty that this attitude was voluntarily checked. But involuntary immobilization in an attitude involuntarily assumed is different from the involuntary maintenance of a contraction *voluntarily* initiated, and continued *in spite of voluntary innervation of the antagonists*. Nevertheless, we suspect that in not a few cases of athetosis, especially such as are mild in degree, the observer will find

that tonic innervation is present in a less well marked or less enduring form. Lewandowsky [25] goes so far as to say that the abnormally long maintenance of a given voluntary innervation can be seen in many cases of ordinary hemiplegia, and that the mildest form of this disturbance is seen in Babinski's adiadochokinesis, where the patient is unable to make a succession of quick alternating movements, because he is unable to inhibit quickly enough a given innervation once it is made. Lewandowsky does not consider adiadochokinesis the expression of cerebellar defect merely, but says it is found also in slight cases of hemiplegia.

We do not know that we agree entirely with Lewandowsky in this, but, at any rate, it cannot be denied that mild degrees of tonic innervation occur, perhaps also in ordinary hemiplegia, and that it is impossible to draw a hard and fast line between what is to be considered worth noticing and what is to be neglected. If in this paper we have dealt with well-marked instances, we cannot furnish any criterion or standard by which to judge when an innervation is sufficiently long to be designated a "tonic" innervation. If it is maintained beyond the command to relax, it is certainly pathological. The cases we have quoted in this section are, we think, free from objection, and they show that tonic innervation, in the strict sense, may occur in association with athetosis. It is practically certain that in the first case the lesion is cortical, and in Kaiser's case it may be so. It is certain that athetosis may result from lesions that are essentially cortical (Demange [9]). Where athetosis and tonic innervation are associated, the assumption may be made that the lesion is likely to be cortical, the more so as Wilson has shown that athetosis has for its basis a disturbance of function of the afferent cerebello-thalamo-cortical path, and the only place where that path and the pyramidal path meet is in the cortex.

Pathogenesis and Localizing Value.

The valuable experiments of Sherrington [37] on reciprocal innervation make it extremely likely that certain disturbances of motility may be explained satisfactorily by a disturbance in the proper relation between impulse and inhibition. Reciprocal innervation is readily produceable from the cerebral cortex, but the cortex is not essential to the reaction. Sherrington has shown that it is obtainable by direct excitation of the internal capsule itself, and the reaction there obtained is just as striking. "It is evident," to use his own words, "that the action of arrest may take place in centres which are subcortical." In

case the reader may erroneously imagine that such subcortical centres are constituted by the basal ganglia, he may be informed that we are assured by Dr. Graham Brown that the reaction is obtainable with equal ease from the pyramids in the medulla. Exner [10] inferred from similar observations that the inhibitory phenomena had their chief seat in the spinal mechanisms, though elicited from the cortex, and Sherrington states that "my own inference has been that the seat of inhibition in these reactions from the motor cortex lies probably at the place of confluence of conducting channels in a common path, likely enough at their confluence upon the 'final common path,' the motor neurone, that is at the ultimate synapse."

We are therefore in possession of physiological data which go to show that as a given muscle is innervated, so its antagonists are actively relaxed, and that this reciprocal innervation is carried out in the spinal cord though it may be elicited from the cerebral cortex. This does not exclude the extreme likelihood, as Sherrington says, that in other fields of action one cortical element inhibits another cortical element.

It appears to us feasible to explain the phenomenon of tonic innervation by a disturbance of reciprocal innervation in "willed" movements. Emphasis is laid on the "willed" movements, because it may be remembered that in our Case 3 the phenomenon was typically and solely present on voluntary movement, and not on involuntary movement. As the patient voluntarily contracts his flexors, say, active inhibition of the antagonists, viz., the extensors, takes place; when next he would reverse this action, he is able voluntarily to innervate the extensors, but there is no corresponding inhibition of the flexors. This constitutes the symptom with which we have been concerned, and while, as has been said, we regard it as a disturbance of reciprocal innervation and localize its mechanism in the cord, we are unable in the present state of our knowledge to go further.

But it may be elicited from the cortex. Our three cases are all cerebral; moreover, the site of the tumour in our first two cases, and the position of the lesion in our third case, as deduced by the remarkable similarity of the case to that reported by Goldstein, where the lesion was accurately determined *post mortem*, are practically identical. The maximum disturbance affected the superior frontal gyrus on the right side; and there must have been direct pressure on the adjacent precentral gyrus, as well as on the middle frontal gyrus and the white matter underlying these convolutions. We are well aware of the

unsatisfactory nature of attempts at correlating lesions and symptoms when the morbid agent is a neoplasm ; but in our third case the lesion is vascular, in all probability, and, as already said, by analogy from Goldstein's case we can determine the seat of the disease with accuracy and with less complication from neighbouring disturbances of function.

In a word, there is reason to suppose that the tonic innervation of the left limbs in our patients is associated with impairment of function of the cortico-spinal neurones from a lesion at their cortical end ; either the activity of the affected motor centres is directly interfered with, or, more likely, the stimuli reaching them by short transcortical paths from a hypothetical psychomotor centre placed in the area in front of the precentral gyrus are interfered with.

It may be remarked also that in all the pathologically corroborated cases the symptom was present only on the side opposite to the lesion ; in left hemisphere lesions it was on the right side clinically, and *vice versa*. It cannot be regarded, therefore, as depending on, or indicative of, an impairment of function of the corpus callosum.

We have met with cases of cortical lesions in approximately the same region where the symptom was not observed, and we imagine, therefore, that its appearance represents a stage in the process of interference with the functional activity of the neurones concerned. It will be remembered that in our first two cases the symptom vanished with the removal of the tumour and the onset, for the time, of more definite hemiplegia, and in a previous section we have alluded to its dependence on the comparative integrity of the cortico-spinal tracts. In view of the fact that in our third case the path was open for involuntary or automatic impulses when it was closed, so to speak, for "willed" movements, we may suppose that the lesion is more likely to be such as to produce a transcortical than a directly cortical impairment of function. But in our ignorance of what is meant by volitional as opposed to involuntary innervation our view must remain merely a hypothesis.

Allusion must be made, finally, to the hypothesis advanced by Kleist [22] [23] to explain the phenomenon of tonic innervation of central origin. Kleist imagines a "tonic co-ordinating" apparatus constituted by an afferent and efferent reflex arc ; the former is the cerebello-rubro-thalamo-cortical (frontal) system, and the latter a doubled path—(1) fronto-pontine tract, middle cerebellar peduncle, nucleus dentatus and back to the nucleus ruber, and (2) direct fronto-rubral path. He further postulates an "apparatus for automatic and expression and associated movements," formed by optic thalamus, corpus striatum, and

corpus Luysii; he thinks that the thalamo-frontal and fronto-rubral systems also enter into this apparatus. In his view disturbances of tonus and co-ordination are linked with the former apparatus, while choreo-athetotic movements, impairment of movements of expression, and associated movements, are linked to the latter.

Apart from other considerations, the uncertainty of the anatomical facts in reference to Kleist's second system make it impossible for us to subscribe to his views, which are, for that matter, contradicted by what is known of the pathological basis underlying both chorea and athetosis.

In regard to the first system, Kleist's hypothesis does not, we think, correspond to the facts of physiology to which allusion has already been made, and on which *inter alia* we rely in our explanation of the nature of the phenomenon known as tonic innervation. A spinal mechanism is at fault, and it is thrown out of gear by defective impulses from the cortex. As for the possibility of the fronto-pontine cerebellar or fronto-rubral system being concerned with co-ordination or the maintenance of tonus, it does not concern the subject of this paper.

BIBLIOGRAPHY.

[1] Batten and Gibb. "Myotonia Atrophica," *Brain*, 1909, vol. xxxii, p. 187.

[2] Bonhoeffer. "Casuistische Beiträge zur Aphasielehre," *Arch. f. Psych.*, 1903, Bd. xxxiv, S. 564.

[3] Bramwell and Addis. "Myotonia Atrophica," *Edinburgh Med. Journ.*, 1913, July, p. 21.

[4] Bremer and Carson. "A Case of Brain Tumour (Angioma Cavernosum) causing Spastic Paralysis and Attacks of Tonic Spasms," *Amer. Journ. Med. Sci.*, 1890, September, vol. c, p. 219.

[5] Breukink. "Ueber Patienten mit Perseveration und asymbolischen und aphasischen Erscheinungen," *Journ. f. Psych. u. Neur.*, 1907, Bd. ix, S. 113.

[6] Campbell, Macfie. "Agraphia in a Case of Frontal Tumour," *Review of Neur. and Psych.*, 1911, vol. ix, p. 287.

[7] Coriat. "The Psychopathology of Apraxia," *Amer. Journ. of Psych.*, 1911, January, vol. xxii, p. 65.

[8] Curschmann. "Ueber partielle Myotonie," *Berliner klin. Wochenschr.*, 1905, Bd. xlii, S. 1175.

[9] Demange. "Contributions à l'étude des tremblements prae- et post-hémiplégiques," *Revue de Médecine*, 1883, May, vol. iii, p. 375.

[10] Exner. "Zur Kenntniss von der Wechselwirking der Erregungen im Centralnervensystem," *Pflüger's Archiv*, 1882, Bd. xxviii, S. 487.

[11] Frohmann. "Ueber einen Fall von Myelitis transversa mit Muskelwogen und eigentümlichen Veränderungen der elektrischen Reaktion," *Deutsch. Arch. f. klin. Med.*, 1906, Bd. lxxxvi, S. 339.

[12] Goldstein. "Zur Lehre von der motorischen Apraxie," *Journ. f. Psych. u. Neurol.*, 1908, Bd. xi, Hft. 4/5, S. 169.

[13] *Idem.* "Der makroskopische Hirnbefund in meinem Falle von linksseitiger motorischer Apraxie," *Neurolog. Centralbl.*, 1909, Bd. xxviii, S. 898.

[14] Gregor and Schilder. "Zur Theorie der Myotonie," *Neurolog. Centralbl.*, 1913, Bd. xxxii, S. 85.
[15] Handelsman. "Ein Fall von Syringomyelie mit neuro- und myotonischer Reaktion," *Neurolog. Centralbl.*, 1911, Bd. xxx, S. 418.
[16] Hartmann. "Beiträge zur Apraxielehre," *Monatssch. f. Psych. u. Neur.*, 1907, Bd. xxi, S. 97.
[17] Hirschfeld. "Myotonia Atrophica," *Zeitschr. f. d. ges. Neurol. u. Psych.*, 1911, Bd. v, Hft. 5, S. 682.
[18] James. "Principles of Psychology."
[19] Kaiser. "Myotonische Störungen bei Athetose," *Neurolog. Centralbl.*, 1897, Bd. xvi, S. 674.
[20] Kleist. "Kortikale (innervatorische) Apraxie," *Jahrb. f. Psych. u. Neur.*, 1907, Bd. xxviii, Hft. 1, S. 46.
[21] *Idem.* "Ueber nachdauernde Muskelkontraktionen," *Journ. f. Psych. u. Neur.*, 1908, Bd. x, S. 95.
[22] *Idem.* "Weitere Untersuchungen an Geisteskranken mit psychomotorischen Störungen." Leipzig, 1909.
[23] *Idem.* "Der Gang und der gegenwärtige Stand der Apraxieforschung," *Ergebnisse der Neur. u. Psych.*, 1911, Bd. i, Hft. 1/2, S. 343.
[24] Kroll. "Beiträge zum Studium der Apraxie," *Zeitschr. f. d. ges. Neur. u. Psych.*, 1910, Bd. xi, Hft. 2, S. 315.
[25] Lewandowsky. "Bemerkungen über die hemiplegische Contractur," *Deutsch. Zeitschr. f. Nervenheilk.*, 1905, Bd. xxix, S. 208.
[26] Liepmann. "Der weitere Krankheitsverlauf bei dem einseitig Apraktischen und der Gehirnbefund auf Grund von Serienschnitten," *Monatsschr. f. Psych. u. Neur.*, 1905, Bd. xvi, S. 289.
[27] *Idem.* "Ueber Störungen des Handelns bei Gehirnkranken." Berlin, 1905.
[28] *Idem.* "Drei Aufsätze aus dem Apraxiegebiet." Berlin, 1908.
[29] Mills. "Myotonia and Athetoid Spasm," *International Clinics*, 1891, April, vol. i, p. 247.
[30] Neisser. "65 Sitzung des Vereins Ostdeutschen Irrenärzte zu Breslau," *Allg. Zeitsch. f. Psych.*, 1895, Bd. li, S. 1016.
[31] Oppenheim and Siemerling. "Ueber das Vorkommen von Hypertrophie den Primitivfasern in Muskelpartikeln, welche dem lebenden Menschen excidirt wurden," *Centralbl. f. d. med. Wissensch.*, 1889, September 28, Bd. xxvii, S. 705.
[32] Pick. "Etude clinique sur les troubles de la conscience dans l'état postepileptique," *Annales médico-psychologiques*, 1903, Jan.-Fév., vol. xvii, p. 18.
[33] *Idem.* "Studien über motorische Apraxie." Leipzig and Wien, 1905.
[34] Piper. "Elektrophysiologie menschlicher Muskeln," Berlin, 1912.
[35] Rhein. "A Case of Apraxia, with Autopsy," *Journ. of Nerv. and Ment. Dis.*, 1908, October, vol. xxxv, p. 619.
[36] Schlesinger. "Die Syringomyelie," Wien, 1902.
[37] Sherrington. "The Integrative Action of the Nervous System," London, 1906.
[38] von Sölder. "Ueber Perseveration, eine formale Störung im Vorstellungsablaufe," *Jahrbücher f. Psych. u. Neur.*, 1899, Bd. xviii, S. 490.
[39] Steinert. "Cerebrale Muskelatrophie, nebst einem Beitrag zur Casuistik der Balkentumoren," *Deutsch. Zeitschr. f. Nervenheilk.*, 1903, Bd. xxiv, S. 1.
[40] Stoddart. "Mind and its Disorders," London, 1908.
[41] van Vleuten. "Linksseitige motorische Apraxie," *Allg. Zeitsch. f. Psychiat.*, 1907, Bd. lxiv, S. 203.
[42] Wilson, S. A. K. "A Contribution to the Study of Apraxia," *Brain*, 1908, vol. xxxi, p. 164.
[43] *Idem.* "Progressive Lenticular Degeneration," *Brain*, 1912, vol. xxxiv, p. 295.

DYSSYNERGIA CEREBELLARIS PROGRESSIVA—A CHRONIC PROGRESSIVE FORM OF CEREBELLAR TREMOR.[1]

BY J. RAMSAY HUNT, M.D.
New York.

PAGE

INTRODUCTION 247
CHAPTER I.—CASE REPORTS 248
CHAPTER II.—ANALYSIS OF THE SYMPTOMATOLOGY 259
The Tremor 259
Dyssynergia and Dysmetria 261
Adiadokokinesis 262
Intermittent Asthenia 262
Hypotonia 263
CHAPTER III.—DIFFERENTIAL DIAGNOSIS 264
CHAPTER IV.—THE RELATION OF THE SYMPTOMATOLOGY TO THE CEREBELLAR MECHANISM 265
SUMMARY 267

INTRODUCTION.

As *dyssynergia cerebellaris progressiva*, I would direct attention to a chronic progressive tremor disturbance, which seems deserving of differentiation as a definite clinical type of nervous disease.

This affection is characterized by generalized intention tremors, which begin as a local manifestation and then gradually involve in varying degree the entire voluntary muscular system. The tremor which is extreme when the muscles are in action, ceases entirely during relaxation and rest. If this disorder of motility is subjected to more detailed study, it will be found that, associated with the tremor, there is a well-marked disturbance of muscle-tone and of the ability to measure correctly direct and associated muscular movements; the clinical manifestations of this are *dyssynergia*, *dysmetria*, *hypotonia*, *adiadokokinesis* and *asthenia*. All of these symptoms, including the volitional tremor, which is only the extreme expression of the underlying disturbance of muscle-tone and synergy, indicate a disorder of cerebellar function.

[1] Presented at a meeting of the American Neurological Association, May 5, 1914. A Preliminary Report to the New York Neurological Society, April 7, 1914.

I, therefore, regard this affection with its progressive tendency, chronic course and well-defined cerebellar symptomatology, as an organic disease caused by degeneration of certain special structures of the cerebellar mechanism, which are concerned in the regulation of the tonus and synergies of muscles.

These cases are further distinguished by the absence of true nystagmus, objective vertigo ("Drehschwindel"), cerebellar fits, vestibular seizures and disturbances of equilibrium, symptoms which are so frequently associated with gross lesions of the cerebellum.

The clinical picture is, therefore, strictly limited to a progressive disturbance of synergic control, the most striking characteristic of which is the ataxic intention tremor, which accompanies any movement of the affected part, whether volitional, reflex or automatic.

Chapter I.—Report of Cases.[1]

Case 1.—*A woman, aged 47. Onset at the age of 40, with volitional tremor of the left arm. One year later similar involvement of the right arm, followed by gradual extension to the muscles of the head, trunk and lower extremities. The clinical picture is one of generalized, coarse, ataxic tremor on attempting any movement, however slight, which ceases during rest. A study of the motility shows also a disturbance of the cerebellar function, viz., dysmetria, dyssynergia, hypotonia, adiadokokinesis and intermittent asthenia; otherwise the neurological examination is negative. The chief symptom is generalized dyssynergia with tremor movements on intention.*

History.—The patient is a woman, aged 47, born in Germany, of Jewish parentage, who was admitted to the Montefiore Home, in August, 1908, since which time she has been under continuous observation.

Family history.—Her father died at the age of 84; her mother is 83, and still living. The parents were not related. One of her brothers died of "scrofula" at the age of 17, and another brother and a sister died in infancy of unknown causes. No members of her immediate family have been subject to tremors of any kind, nor is there any history of such tendency in the collateral branches.

Previous history.—The menses appeared at 13½ years, were regular and of normal character. Menopause at the age of 40. She married at 24, and was never pregnant. For fifteen years she has been a widow. Previous to her marriage she had been a cook, and this occupation she resumed after her husband's death.

Her only previous illnesses have been an attack of enteritis in the summer

[1] From the Neurological Wards of the Montefiore Home and Hospital.

of 1899, influenza in 1890, and pleurisy in 1892. Otherwise she has been well and strong and in excellent health. She was always moderate in the use of alcohol, as well as tea and coffee. There is no history of mental or physical trauma or of venereal disease. For eighteen years she has had a small, firm enlargement of the isthmus and right lobe of the thyroid gland (without symptoms of Graves' disease).

Present illness.—Onset of the disease seven years ago, with an awkward tremor movement of the left hand on attempting to grasp an object. The intensity of this gradually increased, so that with every effort to move the arm there was an awkward shaking tremor. When the arm was at rest there was no tremor. For an entire year this symptom remained limited to the left arm. At the expiration of this time, the same disturbance of motility (volitional tremor) developed in the right arm, and this in turn gradually increased in severity. In the course of the next year a similar shaking and tremor made its appearance in the head and then gradually extended to the trunk and lower extremities. The speech also became affected.

This peculiar motor agitation has shown slow and steady progress, involving in turn the upper and lower extremities, the head and trunk and the muscles of articulation.

The left arm was first affected, a year later the right arm, a few months after this the head and neck and muscles of articulation; after this there was a steady progressive involvement of the trunk and lower extremities, together with an increase in the general severity of the entire tremor disturbance.

During this period of progression, and occasionally since, she has suffered from headaches in the frontal region, but these were never very severe and have not been frequent. They were not accompanied by nausea or vomiting. There has been no diplopia and no attacks of objective vertigo ("Drehschwindel"). A subjective sensation of dizziness and vertigo is not uncommon, especially when the head tremor is excessive. There were never attacks of spontaneous vertigo in the recumbent posture. She has never had any fainting or convulsive seizures.

There is no subjective disturbance of vision, other than that which would naturally accompany the constant nodding and shaking of the head. There have been no paresthesiæ, girdle sensations, and no vesical disturbances. There is no tendency to undue hilarity or exaltation, no explosive emotional attacks and no especial depression. Memory is good and the mentality shows no deterioration or change which is worthy of special mention.

She complains of fatigue and dull aching pains in the muscles of the extremities after exertion; but has never been subject to sharp or lancinating pain.

As a rule she sleeps well, and during sleep all trace of the tremor disappears. When any movements in the bed are made, such as a sudden start or turning, the tremor appears and wakes her; when there is any noise or disturbance in the ward she often wakes in tremor.

The tremor.—If the patient is lying in the recumbent posture with the

body completely relaxed and the head supported by a soft pillow, there is no vestige of any movement.

The slightest attempt at innervation, such as fixation of the eyes, a movement of the hand, a simple flexion of the extremities, or even attempts to speak or smile, are sufficient to produce tremor which is usually increased by the patient's attempt at repression. Mental excitement and effort are also effective in aggravating the tremor. The automatic act of respiration alone, when the patient is quiet and relaxed, does not produce a tremor; during the more violent exacerbations of tremor the respiratory movements are sometimes jerky and arrhythmical.

The favourite position of the patient while sitting is leaning forward, resting the head, arms and upper portion of the body on a table. In this position she will often remain for long periods perfectly quiet and relaxed, unless she is questioned or her attention attracted, when immediately more or less violent tremors result.

While sitting quietly in a chair before the examiner with the arms resting on the lap, the tremor may be confined to nodding and shaking of the head, some facial movements and oscillations of the body; but mental excitement or a slight attempt at voluntary innervation, such as speech or movements of the fingers, seem to disturb the balance and adjustment of the patient, and violent tremors result.

There is no true nystagmus. If, however, the tremor is checked by holding the head, tremor will occasionally appear in the eyes. Such oscillations, however, are not obtained by fixation of an object with the eyes. The overflow of tremor also takes place if the movements of an upper or lower extremity are forcibly checked.

The tremor of the extremities is of the volitional or intention type, and consists of a coarse ataxic shaking and tossing of the extremities rather than a true rhythmical tremor, although in certain positions this ataxic shaking assumes a more or less rhythmical character. It reaches its highest degree of intensity in the upper extremities which are sometimes hurled and thrown about with such violence while under innervation, that severe bruises and contusions may result.

On attempting to place the index finger upon the tip of the nose, the arm is jerked and thrown about with ataxic violence, the motor agitation subsiding, and becoming less severe when the object of the movement is finally reached. On attempting to place the heel upon the knee in the recumbent posture, the same coarse volition disturbances appear; and if the leg is elevated, as with the arm, a violent ataxic tremor develops. Closure of the eyes has no appreciable influence upon the extent or character of the movements.

On standing, the general tremor is much increased, the legs shake, the trunk oscillates, the head is in constant movement, and the arms are tossed and thrown about in most bizarre fashion. Because of the severity of the motor disorder the patient receives all her food and drink from the hands of a nurse. This has been necessary for the past three years. Chewing and

swallowing aggravate the tremor, which adds still more to the difficulty and embarrassment in taking nourishment.

Static equilibrium is well maintained even on a narrow base, and closure of the eyes in this position has no apparent effect upon posture or the intensity and character of the tremor.

For some years all finer movements of the hands have been impossible, and for several years the handwriting has been reduced to illegible scrawls and scratches.

If the patient has had an exciting or fatiguing day, some after-tremor may persist for several hours, even during the period of rest.

On several occasions, during the years of observation in the hospital paroxysms and crises of tremor have occurred, lasting for weeks at a time, in which all of the tremor symptoms have been greatly exaggerated. During this crisis of tremor, standing and walking were difficult and precarious, because of the violence of the motor agitation. After a time, however, the crisis diminishes, equilibrium is restored, and there is a return to the original degree of disability which is chronic and permanent.

The tremor is usually more severe in cool weather.

The speech is slow and scanning and is frequently broken and interrupted by violent explosive efforts and utterances. Under excitement these brusque explosive discharges render it almost unintelligible. The speech disturbance is evidently caused by the same disharmony which characterizes the other muscular efforts. During the act of articulation there are associated tremor-like contractions of the facial movements, and the tremor of the head is much exaggerated.

Asthenia.—There is no paralysis of the muscular system in the usual sense of the word, and the initial muscular effort is carried out with approximately the normal degree of force. There is, however, a curious asthenic symptom of an intermittent character. This is characterized by the inability to sustain or fix a muscular contraction except for short periods of time. If, for instance, the patient is instructed to grasp the hand of the examiner and to maintain the grasp, it is found that the initial effort is of normal force, but after 5 to 10 seconds the grasp relaxes in spite of every effort to maintain the contraction. Immediately after the relaxation of the hand, which is involuntary, another effort is again made, only to suffer the same spontaneous relaxation; so that instead of a single sustained contraction, we are confronted with a series of brief, intermittent muscular contractions of fair intensity. This inability to sustain a muscular contraction, I would designate *intermittent asthenia*, as the clinical impression produced is that of asthenia, although it is not improbable that the underlying disturbance is very closely related to the dyssynergia which will be referred to later.

The intermittent asthenia may also be demonstrated in other muscles of the extremities, as in flexion and extension of the wrist and elbow, abduction of the arms and in the various movements of the legs. It is therefore, like the tremor, a generalized manifestation.

On a few occasions, the patient has suddenly fallen to the ground, because of the giving way of one of the legs when attempting to stand in one position, as before a looking-glass in preparation of her toilet. Such falls are apparently due to an intermittent asthenia of the lower extremities, and are unaccompanied by vertigo or obscuration of consciousness. The electrical reactions of the muscles are normal, both quantitatively and qualitatively. It is interesting to note that strong faradic currents produce sustained muscular contractions without the intermittent relaxation which accompanies voluntary movements.

Hypotonia.—The muscles are well developed and free from atrophy. They are, however, soft and flabby to the feel and there is present a definite hypotonia. The joints are relaxed and flaccid and may be over-extended. This is present in both the upper and lower extremities, and especially in the arms. If the arms are watched during the volitional tremors, it will be seen that, especially in the movements of the hands and fingers, extreme attitudes indicating hypotonia are assumed.

The Stewart-Holmes sign of hypotonia is also present in the upper extremities, i.e., the failure of rebound or recoil when flexion of the arm is resisted and suddenly relaxed. The flexion movement continues until mechanically checked without the intercurrent contraction of the antagonistic triceps. The mechanical irritability of the muscles on percussion is normal: no myotonic phenomena are present.

Dysmetria and dyssynergia. — When the individual movements of the extremities are carefully analysed, a distinct disturbance of the ability to measure, regulate and harmonize voluntary movements is found. In order to eliminate the tremor as far as possible, so that isolated muscular movements may be studied, it is best that the patient be placed in the recumbent posture with the arms relaxed and the head supported. In this position complete motor relaxation and quiet may be produced. If the patient is instructed, with the arm resting on the bed or upon a table, to elevate the index finger and then allow it to fall and to continue repeating this single movement, a number of interesting phenomena become apparent. These tests may also be made with the patient sitting in a chair with the arm relaxed and resting on a table.

This test movement of the index finger is produced by the contraction of the extensor indicis, which is immediately relaxed. Instead of the normal elevation and fall of the finger as should occur, an overaction is observed. The index finger is thrown brusquely as high as possible and remains fixed, relaxation not taking place immediately. Or instead of a single extension movement there may be two or sometimes three successive attempts before the finger is brought into extension. It will also be observed that instead of allowing the finger to fall by simple cessation of contraction, there is an overaction of the antagonists and the finger is thrown down, striking the table with some force.

If the forearm of the patient is encircled by the examiner's hand during these efforts, it will be found that there is a synchronous contraction of the

flexors (antagonists) of the forearm and also occasionally in the muscles of the upper arm.

These symptoms of dysmetria and dyssynergia, which may be demonstrated with ease in the contractions of single muscles, are also very evident in the larger and more complicated movements of both the upper and lower extremities, but may be overlooked and masked by the general tremor which is produced.

Adiadokokinesis was distinctly present in both upper extremities. On attempting to perform quick alternating movements of supination and pronation the movements are slow and interrupted, and there is an inability properly to control and measure them, so that a quick rhythm of normal rapidity is impossible.

This disturbance may also be demonstrated in the movements of the index finger and thumb, preferably tested while the arm is relaxed and resting upon a table.

Sensation.—The general sensations, both superficial (touch, pain and temperature) and deep (muscular and articular), are entirely normal. There is no demonstrable defect in the ability to distinguish the relative difference of weights placed in the hands.

Vision, the sense of smell, taste and hearing are normal and equal on the two sides.

The Bárány rotation and caloric tests show the normal nystagmus reactions on both sides. These reactions are delayed and are obtained with some difficulty, requiring strong stimuli for their production. This is probably due to a certain diminished sensitiveness of the peripheral apparatus of equilibrium by reason of the constant violent oscillations of the head.

The pointing tests ("Vorbeizeigen") are difficult of interpretation because of the violence of the volitional tremor.

Reflexes.—The tendon reflexes of the upper extremities (supinator biceps and triceps jerks) are present, not exaggerated, and are equal on the two sides. The jaw-jerk is present and not exaggerated. The knee-jerk and ankle-jerk are present on both sides and are of equal intensity and not exaggerated. The abdominal reflexes are present and equal. The plantar reflex gives a normal flexor response on both sides, and the Babinski reflex has not been demonstrable during the many years of observation in the hospital.

Cranial nerves.—The pupils are equal and react promptly to light and accommodation; the pupillary skin reflexes are normal.

Ophthalmoscopic examination shows normal optic nerves: no signs of neuritis or pallor of the disc. The ocular excursions are normal, no true nystagmus. The innervation of the facial muscles, the muscles of mastication, soft palate and tongue is normal, but produces marked tremor disturbances.

General examination.—The apex beat is in the fifth interspace within the nipple line. There is a systolic murmur over the body of the heart; the second sounds are not accentuated. Percussion of the lungs and the breath sounds are normal. Percussion and palpation of the abdomen are

negative. There is a slight, firm enlargement of the isthmus and right lobe of the thyroid gland without pulsation or bruit, tachycardia, exophthalmus or other symptoms of Graves' disease. The urine is normal.

The blood contained 4,720,000 red cells; 62,000 white cells; hæmoglobin, 88 per cent. Wassermann tests of blood and cerebrospinal fluid are negative. There was no increase of cells or globulin in the cerebrospinal fluid. There is a marked dermographia, but no pigmentation of the skin or cornea. The gynæcological examination showed no abnormality.

CASE 2.—*A woman, aged 29; onset at the age of 23, with volitional tremor of the left leg. Two years later the left arm showed similar involvement: one and a half years after this the right arm became affected. Since then increase in severity and gradual extension to the head, trunk and right lower extremity. With the tremor are the associated symptoms of a cerebellar disorder, dyssynergia, dysmetria, adiadokokinesis, hypotonia, and intermittent asthenia: otherwise the neurological examination is negative. The clinical picture is one of generalized intention tremors which cease when the muscles are not in action.*

History.—Patient is a married woman, aged 29, of Jewish parentage, who has been under my personal observation for the past year and a half.

Family history.—Her father died of tuberculosis at the age of 45; her mother is still living and is in good health; two sisters and one brother are living and well; two brothers died in early life of unknown causes. There is no history of tremor in the family.

Previous history.—Menses began at the age of 13, are regular but somewhat painful. She married at the age of 24, and has been twice pregnant, bearing healthy children; she has had no miscarriages.

She had measles, diphtheria and scarlet fever before the age of 9 years. With these exceptions she has had no illness and has been in good health until the onset of the present disease. She does not take alcohol and is moderate in the use of tea and coffee. There is no history of luetic infection, physical trauma or mental shock.

Present illness.—The affection from which she now suffers first made its appearance six years ago with tremor of the left leg. On standing or walking the leg is subject to jerky, irregular movements, which interfere with the free-and-easy motion of the extremity. There is no paralysis, no paræsthesiæ, and no pain; when the leg is at rest or in the recumbent posture all tremors cease. Occasionally, however, after fatigue and excitement some tremor persists in the ankle-joint for a short time.

For two years the disturbance was confined to the left lower extremity, gradually increasing in severity. At the expiration of this time the left arm began to show some involvement. Any attempt to grasp an object was accompanied by irregular awkward movements, which ceased as soon as the arm was placed at rest.

One and a half years later a similar tremor appeared in the right arm and has gradually increased in severity.

During the past year the head and trunk show evidences of tremor, and the speech is slow and slightly dysarthric. There is also some tremor in the right leg, so that at the present time there is a generalized intention tremor which affects both upper and the left lower extremity and in a lesser degree the other voluntary movements.

For the past three years all finer movements of the hands have been impossible. It is very difficult for her to eat and drink, and the handwriting is an illegible scrawl. She has at times been able to do coarse work about the house, such as required no special accuracy of movement.

During the course of this affection she has suffered from occasional headaches, chiefly frontal. These occur especially during the menstrual periods. She has had no obvious attacks of vertigo and no diplopia. Occasionally when the head tremor is most severe a sensation has been present of giddiness and lightness in the head. She has had no epileptiform or fainting attacks. The sleep is fair and is undisturbed by any tremor. There are no sphincter disturbances. Her memory is good and there are no evidences of mental deterioration; no emotional crises, and no attacks of forced laughing or crying.

The symptoms have been strictly limited to a chronic, coarse tremor of the intention type, beginning in the left leg and gradually extending to the other extremities, including the head and trunk. This tremor produces some disturbances of gait and station, a marked limitation of the use of the arms, more or less constant nodding and oscillation of the head when held erect, and a slight difficulty in articulation.

The tremor.—If the patient is at rest in the recumbent position, with the head supported and the arms and legs completely relaxed, so that all voluntary muscular innervation is eliminated, there is no sign of tremor. The muscles of the extremities are in a state of perfect rest and quiet. The only exception to this rule is following periods of mental excitement and physical exertion, when some after-tremor may persist for a time; but this passes away if the rest is prolonged. Any attempt, however slight, to carry out voluntary movements—as, for example, fixation of an object with the eyes, a movement of the hand or leg—causes an immediate disturbance of muscle equilibrium, and coarse tremor movements appear in the corresponding region of the body.

In the erect posture, if the patient stands upon a narrow base, there is no sign of ataxia with the eyes either opened or closed. There is simply a nodding and shaking tremor of the head with some oscillation of the trunk and more or less coarse tremulousness of the arms and legs. In walking there is no ataxia in the ordinary sense, but the gait is jerky, uneven and the rhythm and harmony of movement is disturbed by the coarse tremor.

If an attempt is made to place the index finger upon the tip of the nose, the arms are immediately thrown into the violent motor disorder which characterizes the intention tremor. The same is true of the left leg, and to a less extent of the right leg when the heel is carried upon the knee of the opposite leg.

If the arm or leg is elevated, the extremity is immediately thrown into a violent atactiform shaking tremor, which ceases as soon as the voluntary

innervation is removed and the part is placed at rest. Even slight voluntary movements, as of the fingers or hand, tend to produce considerable motor disorder of the whole extremity, unless the voluntary innervation is checked and the part placed at rest.

The speech is slower than normal and slightly scanning and at times uneven and slightly dysarthric. Mastication and deglutition are performed without difficulty, but are associated with an increase of the head tremor. There is no tremor of the facial muscles.

Asthenia.—The gross motor power of the extremities is undisturbed. There is, however, an intermittent asthenia or inability to sustain, except for brief periods of time, a co-ordinated muscular movement. For example, on attempting to maintain a hand clasp, in spite of every effort the grasp will relax and another attempt must be made, which in turn is only of short duration; so that instead of a single sustained contraction of the muscles concerned in maintaining a grip of the hand, there occurs a succession of interrupted efforts towards the same object. This may also be demonstrated in flexion of the elbow, extension of the foot or any other voluntary movements in which such a test could be carried out.

The electric excitability of the muscles to both faradic and galvanic currents shows no deviations from the normal. With strong faradic currents, tonic contractions of the muscles may be produced without the intermittent relaxation noticed in voluntary effort.

Hypotonia.—The muscles are fairly well developed and show no signs of atrophy. They are, however, soft and flabby on palpation and the joints are relaxed and flaccid. This is especially true of the upper extremities, in which the Stewart-Holmes sign of hypotonia is present. Mechanical irritability of the muscles is normal on percussion.

Dyssynergia and dysmetria.—If the arm is placed in a relaxed position and the muscular contractions of single movements are studied, there will be found evidences of dyssynergia and dysmetria. This is even more apparent in an analysis of the more complicated co-ordinated movements of the extremities, although somewhat masked and less readily identified because of the marked generalized tremor disturbance which results.

Adiadokokinesis is present on both sides.

Sensation.—Superficial and deep sensibility are entirely normal. There is no demonstrable defect in the ability to determine the relative difference of weights placed in the hands.

Vision, smell and hearing are normal. The Bárány rotation and caloric tests produce the normal nystagmus reactions, showing the integrity of the labyrinthine apparatus on both sides. The pointing tests are difficult of interpretation because of the coarse tremor.

Reflexes.—The supinator, triceps and biceps jerks are present, not exaggerated, and are equal on the two sides. The jaw-jerk is present and not exaggerated. The knee-jerks are active, slightly exaggerated and are of equal intensity on the two sides. The Achillis jerks are present and equal on the

two sides. There is no patellar clonus and no ankle clonus. Frequent examinations have shown that the abdominal reflexes are constantly present and equal on the two sides. Plantar stimulation produces normal flexion of the toes. (No Babinski.)

Cranial nerves.—The pupils are equal and react promptly to light and accommodation; the pupillary skin reflexes are present. The ocular excursions are normal; no nystagmus. Ophthalmoscopic examination of the optic nerves is negative. There is no pallor of the disc and no sign of neuritis. Innervation of the face, muscles of mastication, soft palate and tongue are normal.

General examination.—There is a moderate degree of dermographia; no pigmentation of the skin or cornea. The heart and lungs are normal; urine is normal.

Wassermann tests of the blood and of the cerebrospinal fluid are negative. There is no increase of the cellular elements or of the globulin content of the cerebrospinal fluid. Abdominal palpation and percussion are negative. No enlargement of the thyroid gland.

Gynæcological examination shows multiple stellate lacerations of the cervix, anteversion of the uterus and a mass in the right broad ligament with thickening of the Fallopian tube. The ovaries are normal.

CASE 3.—*Onset of the disease in a man, aged* 28, *with intention tremor of the right arm, followed six months later by a similar disturbance of the left upper extremity: gradual extension to the head and muscles of articulation. Slight involvement of the lower extremities; otherwise the neurological examination is negative. The clinical picture consists of intention tremor of the upper extremities, with involvement of the head, speech, and in a lesser degree of the legs. In the upper extremities, dyssynergia, dysmetria, adiadokokinesis, hypotonia and intermittent asthenia are associated with the tremor.*

History.—Patient is a man, aged 31, a native of Bohemia who has lived in the United States for the past six years. He is a labourer by occupation, is married, and has two children living and well. His wife has had no miscarriages. There is no history of tremor in the family, and his parents are still living and in good health. There is no history of venereal disease and no trauma. He is unable to give any satisfactory data as to the diseases of early childhood, but since that time he has always enjoyed robust health and has had no serious illnesses.

Present illness.—The disease from which he now suffers made its appearance a little over three years ago, with a shaking tremor of the right hand on attempting any movement. This gradually increased in severity; six months later a similar tremor made its appearance in the left arm and gradually progressed. Following this, oscillations of the head appeared while sitting or standing, together with a disturbance of speech. Later the lower extremities became somewhat affected, but in a less degree.

There have been occasionally frontal headaches since the beginning of the tremor, but they are not severe and are unaccompanied by nausea and vomiting.

He has had no vertiginous seizures, and no diplopia. There have been no pains in the extremities, no paræsthesiæ, no vesical disturbance, no attacks of forced laughter or crying, no fainting and no convulsive seizures. Because of the tremor, the man was forced to abandon his occupation, which consisted of general work of a coarse nature. Movements of the arms, as are required in dressing, eating and drinking, are rendered very difficult and almost impossible by reason of the coarse volitional tremor. There are no subjective disturbances of sight or hearing, and apart from the tremor disturbance his general health is good.

Tremor.—In the recumbent posture, with the head supported there is no visible or palpable tremor movement of the head, trunk or extremities. If, however, the arm is elevated there immediately develops a coarse shaking volitional tremor, which continues as long as the innervation is maintained. If the arm is replaced by the side and voluntary innervation is interrupted, all tremor immediately ceases. The same tremor disturbance is present in the left arm and to a lesser extent in the legs.

If, when lying in the recumbent posture the leg is elevated or an attempt is made to place the heel upon the knee, a coarse shaking and volitional tremor makes its appearance. If the forefinger is carried to the nose, violent tremors of the intention type are produced. It is impossible for the patient to carry a glass of water to the lips without forcibly spilling the contents.

On standing there develop nodding and oscillation of the head. There is also a slight palpable tremor of the lower extremities and some oscillation of the trunk in the erect posture. After excitement or prolonged examination some after-tremor may persist in the recumbent posture, but soon passes off with the rest.

The tremor movement is slow, ranging from three to five per second. The rate and amplitude are increased by excitement and effort. The gait is but little disturbed. The patient can walk a fairly straight line, and the chief manifestations during walking are the shaking of the arms and head. There is no tremor of the face or of the eyes: no nystagmus. The speech is slow and scanning in type, and becomes dysarthric and difficult to understand during excitement.

Asthenia.—The gross motor power of the arms and legs is undisturbed. In the upper extremities, however, there is a distinct inability to sustain prolonged muscular contractions (intermittent asthenia). This is not so evident in the lower extremities where the tremor is comparatively slight.

Hypotonia.—The muscles are well developed and show no signs of atrophy. There is, however, some hypotonia of the upper extremities. The joints and muscles are flaccid and relaxed and the Stewart-Holmes sign of hypotonia is present.

Dyssynergia and dysmetria.—If single movements are studied, like those of the extensor indicis, evidences of dyssynergia and dysmetria are demonstrable. These are also present in the larger movements of the upper extremities and to a lesser degree in the lower extremities.

Adiadokokinesis is present in both upper extremities.

Sensation.—Superficial and deep sensibility are normal. Vision, smell and hearing are normal.

Reflexes.—Supinator, biceps and triceps jerks are equal and normal. The knee-jerks are present and equal, not exaggerated. The ankle-jerks are present and equal, and there is no clonus. The cremasteric and abdominal reflexes are present and equal. Plantar stimulation produces flexion of the toes; no Babinski.

Cranial nerves.—Pupils are equal and react promptly to light and accommodation. Ocular excursions are normal; no nystagmus; the optic discs are normal in appearance. Innervation of the soft palate, tongue, and muscles of mastication are normal.

General examination.—Patient is a strong, robust looking man; no pigmentation of the skin or cornea. Heart and lungs are normal; urine is normal. Wassermann tests of the blood and cerebrospinal fluid are negative. No increase of cells or of globulin in the spinal fluid.

Chapter II.—Analysis of the Symptomatology.

General remarks.—The three cases which have been described are similar in their symptoms and clinical course. They differ only in degree and in the duration of the disease.

In all, the symptomatology is limited to a more or less generalized tremor during muscular activity, which is especially severe in the extremities. Associated with and underlying the volitional tremor, are symptoms of dyssynergia, dysmetria, adiadokokinesis, asthenia and hypotonia. In all other respects the neurological examination is negative. There is no nystagmus, no objective vertigo, no disturbances of the static or kinetic equilibrium, and no convulsive seizures. The intellectual functions are intact and the optic nerves are normal. Occasional frontal headaches were observed in all of the cases. These were never severe and were not associated with nausea or vomiting. Subjective sensations of giddiness and vertigo were present at times, especially in Case 1. This was not associated with a feeling of rotation or of movements of external objects, and was apparently caused by the constant shaking of the head. In none of the cases was there a family history of tremor, or evidence of a tremor tendency before the onset of the disease.

The tremor. — The intention tremor is the most striking and characteristic symptom of the disease. This begins in one extremity and progresses slowly, involving gradually and successively the remaining

portions of the body. Three or four years were required for the tremor to become generalized. It consists of a coarse, irregular, atactiform shaking or "Wackeln" on attempting any movement. The tremor movement is slow, ranging from three to five vibrations a second; both the rate and amplitude are increased by mental and physical activity. It ceases entirely in a relaxed or recumbent posture and is consequently absent during sleep. It may happen, if the paroxysms of motor agitation have been prolonged and severe, that some after-tremor may persist, even during rest. This, however, is only observed after unusual efforts or excitement and is of short duration, gradually subsiding with rest and quiet.

If in a relaxed or recumbent posture with the muscular system quiescent, the slightest attempt is made to innervate a muscle, the tremor immediately reappears. It will sometimes happen that the head and extremities are not well supported in the recumbent posture, and some motor perturbation continues even in a state of apparent rest; this persists until the patient is placed in a more favourable position in which the muscular relaxation is complete.

In its severer form, the gait is affected and becomes jerky and uneven, the head is in constant and violent oscillation, the face tremulous, speech scanning and explosive, and the arms are tossed and thrown about in a most bizarre and random manner. In this state of general motor agitation the appearance of Huntington's chorea is more or less suggested, except that the character of the movements is irregularly tremulous rather than choreiform. There is no true nystagmus on fixation of an object with the eyes. If, however, the tremulous shaking head is firmly held and the tremor thus checked, it may overflow and reappear as an oscillation of the ocular movements. At times there are spontaneous rhythmical and rolling movements of the eyes. These may also occur on fixation of an object and are related to the general motor disturbance, and have not the character or constancy of true nystagmus.

There are days, and sometimes longer periods of even weeks, during which the tremor suffers temporary exacerbations. After the subsidence of these crises of tremor there is a return to the usual chronic condition. The whole course of the disease is chronic and slowly progressive, and the motor life becomes more and more restricted; so that the patients in time are almost entirely dependent upon the care of nurses or relatives. Once established, the tremor never disappears, except during rest. In one patient it was exaggerated by cold.

Those muscles which are concerned in the performance of certain automatic functions, like the diaphragm and intercostals, show but slight involvement, and this only during paroxysms of tremor. On such occasions, jerky respiratory movements are not infrequent; at rest in a recumbent position the breathing is regular and uninterrupted. The large muscles of the trunk are but little affected in comparison with the extremities; this is shown by oscillations and tremulous movements of the body in the sitting and erect posture, and by occasional contractions and stiffening of the abdominal musculature.

The tremor is therefore greatest where the voluntary muscular activity has reached its highest degree of functional development and differentiation, as in the upper and lower extremities, and in the head and neck.

The dyssynergia and dysmetria. — The general disturbance of motility is so gross in these cases, that in order properly to study the motor phenomena it is necessary to analyse such movements as require the contraction of a single muscle only or of a small number of muscles. A study of isolated single movements will reveal immediately a difficulty in controlling the measure and association of such movements (dysmetria and dyssynergia). The movement occurs too brusquely or with undue force, so that the aim is over-reached; or it may be insufficient and fall short of the object. This disturbance is usually greater on first efforts or if it be requested that the movement be performed quickly.

Closure of the eyes has no apparent effect upon these motor disturbances, which are very evident in the various segments of both the upper and lower extremities, but which are to a large extent concealed and masked by the tremor.

As a preliminary to performing these tests, the patient should be placed in a recumbent or sitting posture with the arm relaxed and supported by a pillow, preferably with the hand prone. In this position all spontaneous tremor may be eliminated. If the patient is then requested to raise (extend) and then drop the index finger, a disorder of the movement becomes evident at once. In an effort to repeat rapidly the movement of lifting and then letting fall the index finger, it is over-extended and is held fixed in this position of over-extension, and immediate decontraction does not take place; or it may be that two or even three successive attempts are first made before the finger is brought into a position of extension. Then instead of the simple normal relaxation which would allow it to fall almost by its own weight, it is brought down with undue force by a contraction of the flexor muscles. In

other words, there is an inability to carry out quick, rhythmical movements, requiring contractions and relaxations of a very simple kind.

If during these attempts at rhythmical extension of the index finger the muscles of the forearm are encircled by the examiner's hand, it will be observed that with the effort to contract the extensor indicis strong simultaneous contractions of the flexor muscles of the forearm frequently occur, indicating a disturbance in the harmony of the association mechanism of movement (dyssynergia). Such disturbances of the synergy and the measure of movements are even more apparent when larger and more complicated co-ordinated acts are attempted, such as extension of the hand, flexion of the forearm, and the like.

The presence of these disorders of motility gives an insight into the true nature of the tremor, and explains the general motor agitation which is produced whenever the muscular system is thrown into action. With the very evident lack of control in regulating single movements, it does not seem surprising that the patient is utterly at sea in the effort to control the muscles during the activity of an entire extremity. In the effort to control and regulate the muscle synergies under these conditions we have produced the coarse atactiform tremor which is characteristic of the disease.

Adiadokokinesis.—The adiadokokinesis of Babinski is also typically present in these cases. Because of the tendency to general tremors of the arm, this symptom may also be tested to advantage with the arm relaxed and the hand resting on a table. In this position, it is apparent that the power to perform quick successive rhythmical movements is very much diminished. The initial movement is prolonged or insufficient, relaxation does not take place at the proper time, and the succession movements are therefore slow and interrupted. The close relationship of this symptom to the dysmetria and dyssynergia is also very evident.

Intermittent asthenia.—Although the patients tire readily, there is no true paralysis in this group of cases. There is, however, a curious disturbance of the innervation which is best designated as intermittent asthenia. This consists of an inability to sustain or fix muscular contractions except for brief periods of time. For example, if a patient is asked to grasp the hand of the examiner and to maintain the grip firmly, this is found to be impossible. The patient grips the hand firmly at first, and sustains the contraction for a few seconds (five to ten seconds), when spontaneous relaxation takes place. Another attempt is then immediately made only to suffer relaxation again, and even with the greatest effort the contractions cannot be sustained, and are interrupted by spontaneous and involuntary relaxations.

Again, if an attempt is made to hold out the extended arm at right angles, it will be observed that in spite of every effort it tends to fall from the horizontal position and is again thrown back into the horizontal. After this effort has been repeated a few times the arm suddenly falls to the side, the patient apparently being unable any longer to sustain the muscular contractions. After a short period of rest the test may be repeated but with the same result. This inability to sustain contractions may also be demonstrated in flexion movements of the arm, dorsal flexion of the foot, abduction of the arm; in short, any of the movements of the extremities. The efforts to carry out such tests of the motor power are accompanied by considerable fatigue.

In the demonstration of this test, as in those for the dysmetria and dyssynergia, it is preferable that the arm be relaxed and at rest on a table, thus eliminating as far as possible the volitional tremor.

In some respects, this symptom of intermittent asthenia is the reverse of what Babinski has described as cerebellar catalepsy, the ability to fix unduly and immobilize a movement. It is possible that the cerebellar catalepsy is an irritative or spasmodic manifestation while the intermittent asthenia results from the loss of certain cerebellar functions of control.

It is of interest to note, that the intermittent relaxation does not occur when the muscles are contracted independently of the will by strong faradic currents.

Hypotonia.—In the recumbent posture with the muscular system relaxed, palpitation shows the muscles of the extremities to be soft and flabby and the joints unduly relaxed and flexible. This condition of hypotonia is present in both the upper and lower extremities, more especially in the arms. This is by no means so extreme as in certain cases of tabes dorsalis, and while the joints may be unduly extended, the limbs cannot be placed in those abnormal attitudes which is sometimes the case in the severer forms of spinal hypotonia.

The Stewart-Holmes sign of hypotonia in the upper extremities is present: i.e., a failure of the recoil or rebound on resisting a flexion movement of the arm and suddenly releasing it. In performing this test, it is found that the flexion movement of the arm after its sudden release is continued until mechanically checked, there being no reflex contraction of the antagonists, the extensors of the upper arm.

The myotatic irritability of the muscles is unchanged and the tendon reflexes are of normal intensity, showing neither special increase nor diminution.

Chapter III.—Differential Diagnosis.

In general appearance, the motor disturbance which characterizes the progressive cerebellar dyssynergia is similar to the intention tremor of multiple sclerosis. It differs, however, in the slow and gradual manner of progression and the strict limitation of the symptomatology to tremor and the associated dysmetria, dyssynergia, hypotonia, and intermittent asthenia.

All other symptoms of multiple sclerosis, such as nystagmus, objective vertigo, pyramidal and sensory tract symptoms, temporal pallor, hemiplegic attacks, forced laughter, alterations of the reflexes are absent; so that a cerebellar type of this affection may be reasonably excluded.

The pseudo-sclerosis of Westphal may also be eliminated by reason of the strict limitation of the symptomatology to the volitional tremor, together with the absence of mental deterioration, pigmentary deposits and the other symptoms which characterize the recorded cases of this obscure affection.

The theory of a functional disturbance, in the nature of hysteria or the traumatic neurosis, is not tenable in the absence of an adequate etiological factor, and the mental and somatic symptoms which characterize these affections.

The rare tremor type of Parkinson's disease may likewise be excluded by reason of the nature of the tremor and the existing hypotonia and dyssynergia, which differ fundamentally from the muscle manifestations which characterize this affection.

Huntington's chorea, athetosis and myoclonus multiplex are readily differentiated by the character of the motor disorder and persistence during muscular relaxation.

Of especial importance from the diagnostic standpoint is the group of the so-called essential, hereditary or family tremors, the first systematic description of which was given by Dana in 1887. In the original description of this affection the clinical picture was defined by Dana [3] as follows: "The affection in question consists of a fine tremor, constantly present in typical cases during waking hours, voluntarily controlled for a brief time, affecting nearly all the voluntary muscles, chronic, beginning in very early life, not progressive, not shortening life, not accompanied with paralysis or any other disturbances of motor function. It resembles to some extent the tremor of paralysis agitans, still more a simple neurasthenic tremor. It ceases during

sleep, and can be inhibited temporarily by the will. It does not interfere with delicate co-ordination. It neither stops nor increases on ordinary voluntary movements."

At the present time there is an extensive literature treating of this subject (Flatau [4]), the great majority of the recorded cases coinciding in the main with the clinical picture as outlined by Dana. In a few, however, the tremor is described as coarse ataxic and of the intention type, and especially in those cases recorded by Minkowski [6] and Graupner [5] the tremor was gradually progressive and of a severe intention type. The resemblance of such cases to those which are the subject of this study is very striking, and it is not improbable that some of the cases which are now grouped with the hereditary and essential tremors would show on closer examination the same progressive disturbances of the cerebellar function as do the cases which are the subject of this study. It is certainly desirable that the cases of so-called hereditary and essential tremor should be approached from this point of view.

From my own investigations, I am inclined to make a sharp distinction between the hereditary tremor of the type described by Dana and the motor disturbance which is here described as progressive dyssynergia. This is not a true tremor, but a synergic disturbance which is evident only when the extremity is in action, and consists of coarse irregular tremor-like movements in which the constant, vibratory characteristics of the true tremor is almost entirely lacking.

Furthermore, the cases of essential tremor which have come under observation during my investigations of this subject have failed to show the gradual mode of progression, and the associated symptoms of cerebellar disturbance which characterize the dyssynergia.

It may be well to emphasize the fact that in none of my cases of progressive cerebellar tremor was there any hereditary tendency or the slightest indication of tremor before the onset of the disease, which is so common a forerunner of the essential tremor neurosis.

Chapter IV.—The Relation of the Symptomatology to the Cerebellar Mechanism.

The more recent clinical and experimental studies of cerebellar function have shown very clearly that this organ, in addition to the role of maintaining equilibrium, plays an important part in the regulation and control of voluntary movements.

It is this function of the cerebellum which shows evidence of disturbance in dyssynergia progressiva. The absence of nystagmus, objective vertigo, cerebellar fits and disorders of the equilibrium would indicate that the structures subserving those functions are not affected.

The special influence which the cerebellum exercises upon voluntary movements is that of a controlling and reinforcing mechanism which is directly concerned with the regulation of tone, and the direction and measure of movements, the maintenance of attitudes, and the control of the synergies of co-ordinated movements. When these functions are disturbed there result the recognized classical symptoms of cerebellar origin; hypermetria, dysmetria, hypotonia, asthenia, cerebellar ataxia, volitional tremor and asynergia.

All modern authorities agree that the synergic function of the cerebellum is one of special importance. This is the faculty of accomplishing simultaneously and harmoniously the several movements concerned in a co-ordinated act. A disturbance of this function produces asynergia, which is so important a factor in the production of the cerebellar ataxia.

The cerebellar tremor, which is a volitional tremor, is also closely related to the loss of synergic control, and is regarded by most investigators in this field as an expression of asynergia. In other words, cerebellar tremor and cerebellar ataxia may be summed up as a cerebellar asynergia.

Babinski [1] has expressed this view in his brochure published in 1906, as follows:—

"The intention tremor which was first observed in multiple sclerosis is, according to all appearances, due to an alteration of the cerebellar mechanism. It may be explained by a defect in synergy among the different elementary movements of the upper extremity and more particularly in a disharmony in the function of antagonistic muscles."

Stewart and Holmes [9], in a discussion of the symptomatology of cerebellar tumours, record the interesting fact in their remarks upon cerebellar ataxia that "in cases of chronic course, or when the lesion has became latent, the ataxia is as a rule less definite. Then it may be less typical and approximate to the intention type, characteristic of disseminated sclerosis."

My own observations of the progressive cerebellar tremor fully confirm this point of view, namely, that intention tremor is essentially a disturbance of the synergies of muscles, and for this reason

the designation progressive cerebellar dyssynergia was chosen, as best expressing the true nature of the motor disturbance and its relation to the cerebellar mechanism.

If the symptomatology of the group of tremors under discussion is given careful consideration it becomes apparent that in all respects it is identical with the recognized symptomatology of cerebellar disease. There is the same hypermetria, dysmetria, hypotonia, asthenia, asynergia and tremor. All are present in their most exquisite form.

The exact localization of these functions in the cerebellar mechanism is still unknown. It can hardly be doubted, however, from recent experimental, anatomical and clinical investigations, that the cerebellar hemispheres (the "neo-cerebellum") are chiefly concerned with the regulation of the activities of voluntary movements.

Furthermore, the studies of Van Rynberk [8], Bárány [2], and Rothmann [7], have shown that there is a cortical representation in the cerebellar hemispheres, not only of the extremities but also, in all probability, their individual segments and articulations; so that the existence of cerebellar cortical centres for the control of co-ordinated movements is by no means improbable. If these results are confirmed, we have here represented the cortical mechanism for the control of voluntary movements, the loss of which results in these various motor disturbances of cerebellar disease.

In dyssynergia cerebellaris progressiva the clinical symptoms would indicate a progressive disease of that portion of the cerebellar mechanism which is engaged in the regulation of the tone, measure and synergies of co-ordinative movements. It begins gradually, and in the manner of an organic degenerative affection slowly affects the entire mechanism of the cerebellum. Apart from this, there are no other symptoms, the clinical picture is one simply of more or less generalized dyssynergia. For these reasons I believe that in this affection we have to do with a progressive degeneration of certain specialized cell or fibre systems of the cerebellar mechanism, the exact localization of which must await the results of pathological investigation.

Summary.

The results of my investigations may be summarized as follows:—

There exists a chronic progressive form of cerebellar tremor, the most striking and characteristic symptom of which is a generalized volitional tremor which begins locally and gradually progresses.

In its advanced stage the disorder of motility is comparable in severity and violence with that of Huntington's chorea or the generalized athetosis. There is, however, this difference, that in a position of rest and muscular relaxation the tremor movements cease.

An analysis of the motor disorder show a marked disturbance of the ability properly to control and regulate co-ordinated movements. This is shown by the presence of hypermetria, dysmetria, adiadokokinesis, dyssynergia, hypotonia, and intermittent asthenia.

All of these symptoms, including the volitional tremor, coincide with the classical symptomatology which results from a loss of the cerebellar control over voluntary movements. The disorder is, therefore, regarded as of cerebellar origin.

The local onset, gradual progression, and chronic course indicate a progressive degeneration of certain special structures of the cerebellar mechanism presiding over the control and regulation of muscle movements.

Other symptoms of cerebellar disease, such as disturbances of equilibrium, objective vertigo, nystagmus, cerebellar fits and seizures are absent. For this chronic progressive disorder of the cerebellar mechanism the name *dyssynergia cerebellaris progressiva* is suggested as best indicating the essential element in the motor disturbance (dyssynergia), its progressive tendency and relation to the cerebellum. The chronic progressive cerebellar tremor is, however, equally descriptive and may be found preferable.

REFERENCES.

[1] Babinski. "Synergie et inertie cérébelleuse," *Soc. de Neurol.*, July 5, 1906. (*See Archives de Neurol.*, 1906, t. xxii, p. 48.)

[2] Bárány. *Wien. klin. Wochenschr.*, 1912, Bd. xxv, S. 2033.

[3] Dana. "Hereditary Tremor: a hitherto undescribed form of motor neurosis," *Amer. Journ. Med. Sci.*, 1887, xciv, p. 386.

[4] Flatau. "Ueber hereditärem essentiellen Tremor," *Arch. f. Psych.*, 1908, Bd. xliv, S. 306.

[5] Graupner. "Hereditärem Tremor," *Deutsche Arch. f. klin. Med.*, 1899, Bd. lxiv, S. 466.

[6] Minkowski. "Ein Fall von hereditarem Tremor," *Münch. med. Wochenschr.*, 1901, Bd. xlviii, S. 607.

[7] Rothmann. *Berl. Gesellsch. f. Psych. u. Nerv. Krank.*, March 14, 1910. (*See Berl. klin. Wochenschr.*, 1910, Bd. xlvii, S. 1035.)

[8] Van Rynberk. *Folia Neurobiologica*, 1908, Bd. i, S. 403.

[9] Stewart, Grainger, and Holmes, Gordon. "Symptomatology of Cerebellar Tumours," *Brain*, 1904, vol. xxvii, p. 534.

THE PHYSIOLOGICAL SIGNIFICANCE OF THE REFLEX PHENOMENA IN SPASTIC PARALYSIS OF THE LOWER LIMBS.

By F. M. R. WALSHE, M.D., B.Sc., M.R.C.P.

Resident Medical Officer to the National Hospital, Queen Square.

PAGE

INTRODUCTION ... 269

CHAPTER I.—HISTORICAL ... 271

CHAPTER II.—METHODS OF INVESTIGATION ... 274

CHAPTER III.—THE FLEXION REFLEX ... 276

§ 1.—The Receptive Field of the Reflex ... 277

§ 2.—The Nature of the Stimulus ... 279

§ 3.—The Motor Reflex Response. The "Extensor Response" ... 281

§ 4.—Involuntary Flexor Spasms and Paraplegia in Flexion ... 288

§ 5.—Reciprocal Innervation in the Flexion Reflex ... 289

§ 6.—Reflexes accessory to the Flexion Reflex; Crossed and Homolateral Extension; the Crossed Plantar Reflex ... 291

§ 7.—Factors modifying the Reflexes ... 298

(*a*) Muscle Tone in the Limb ... 298

(*b*) Sensory Disturbances in the Limb ... 302

CHAPTER IV.—SUMMARY OF CLINICAL OBSERVATIONS ... 303

CHAPTER V.—PHYSIOLOGICAL CONSIDERATIONS ... 305

§ 1.—The Physiological Significance of the Flexion Reflex and of the "Extensor Response" ... 305

§ 2.—The Dual Nature of the Motor Innervation of the Limb ... 311

§ 3.—Total Lesions of the Cord ... 321

CHAPTER VI.—GENERAL SUMMARY ... 325

APPENDIX I.—Illustrative Cases ... 327

APPENDIX II.—Note on Graphic Methods ... 333

INTRODUCTION.

WHILE the destruction of pyramidal fibres is universally accepted as an adequate explanation of the loss of voluntary power in spastic paralysis, there is still much discussion as to the cause of the other phenomena that are constantly present, namely the hypertonus of the muscles, the exaggeration of the tendon-jerks and the reflex movements that occur.

The original view of Charcot, who regarded spasticity as an

irritation phenomenon, has been universally discarded as plainly untenable, while the illuminating and philosophical conceptions of Hughlings Jackson, to which I shall later have occasion to refer in detail, have not received from modern workers the appreciation or comprehension they would seem to demand if the problems of spastic paralysis are to be solved.

To the German school of neurologists we owe a great number of recent researches into the phenomena of spastic paralysis. Prominent among these are the contributions of Foerster [20], v. Monakow [32], and Rothmann [35]. The hypotheses put forward by these authors deal with hypertonus and contracture rather than with reflex movement. To the French school of neurologists, mainly, we owe our knowledge of the striking reflex movements that occur in the lower limbs in cases of spastic paralysis, but, until quite recently, there has been no attempt to elucidate the physiological significance of these, and it is within the last few years only that, in the work of Marie and Foix, we find a departure among French writers from the merely clinical description of phenomena that have been studied since the days of Charcot.

Although the two last authors have thrown considerable light on the nature of these reflex movements, there is still to be attempted any real effort to correlate them with the other features of spastic paralysis; and it still remains true that many of the phenomena of spastic paralysis are obscure and problematic in origin, and many clinical observations on this subject have yet to find their place in any body of physiological theory. Further, the masterly work of Sherrington has not received from clinicians the attention it merits, and with the exception of two papers of recent date by Marie and Foix has not inspired any clinical work of note; no broad attempt has been made to correlate the physiological findings of Sherrington in animals with the phenomena observed in man in certain conditions of disease of the nervous system.

The observations recorded in this paper have been made with this end in view, and a special study, therefore, of the phenomena of spasticity, contracture, exaggeration of the tendon-jerks and of reflex movements has been made. It is proposed in this present paper to give the results of detailed examination of the reflex movements found in the lower limbs in a large number of cases of spastic paralysis of all kinds, special attention being given to the relation between these and the other above-mentioned features of spastic paralysis. In a subsequent paper it is hoped to deal with the kindred phenomena of hypertonus and contracture.

I wish here to express my indebtedness to the medical staff of the National Hospital for permission to record the results of my examination of cases in the Hospital practice, and to Dr. Hood for placing at my disposal the patients under his care in the St. Marylebone Infirmary. To Dr. Gordon Holmes I am deeply indebted for much advice and assistance.

Chapter I.—Historical.

The reflex movements of the lower limbs in spastic paralysis have long been familiar to neurologists. They include the common involuntary flexor spasms seen in many cases of paraplegia, and the reflex movements occurring on stimulation of the limb, the "Abwehrbeugereflexe" of German authors, the "Réflexes cutanés de défense" of Babinski. We must add to these the more recently described "extensor response"; Babinski's "Phénomène des orteils."

It is not generally recorded in references to the earlier views on the causation of the "positive symptoms" (Jackson [25]) of spastic paralysis, namely the spasticity and reflex augmentation, that Charcot [13] taught that there were two factors responsible for the appearance of these, "the suppression of the moderator influence of the brain, and probably, also, the irritation of which the grey substance, in its turn, becomes the seat."

In regard to the inhibitory function of the pyramidal system Foerster [21] considers the removal of this function is the primary cause of spasticity. When it fails, "the unimpeded sensory afflux charges more and more the anterior horn cell, increases its excitability, and produces a permanent motor stream from the ganglion-cell to the muscle, that is to say, the spastic contracture." To explain the associated reflex movements [22] he assumes that the cerebral control over lower motor mechanisms being lost, the limbs come again under the influence of certain phylogenetically old subcortical motor centres, which determine an attitude of flexion of all the limbs, a "subcortical fixation reflex." Our ancestors, he assumes, were climbing animals, and as such used the flexors to a greater extent than other muscles. In the new conditions, these react more readily to peripheral irritation, and hence we see, for example, the well-known flexor spasms of spastic paraplegia.

V. Monakow [32] and Rothmann [35], although they do not believe in the inhibitory function of the pyramidal tract, agree with

Foerster in ascribing to extrapyramidal subcortical motor systems an important rôle in the causation of spasticity and reflex phenomena.

Babinski regards the reflex movements as defensive in nature. Marie and Foix [30] regard all the reflexes of the spastic lower limb as evidence of spinal function liberated from cerebral control. They accordingly describe them as "Réflexes d'automatisme médullaire."

These authors, and later Strohl [39], describe reflex flexion of the limb as occurring invariably on stimulation of its skin or deep structures, except in a few cases where stimulation of the proximal parts of the limb gives reflex extension. Moreover this flexion is often accompanied by simultaneous extension of the opposite limb. They conclude that these reflex movements of the lower limb represent the mechanism of locomotion, a spinal function liberated from higher control by a lesion of central descending paths. They consider that the movements they have seen clinically in man are analogous with the "spinal stepping" of animals described by Sherrington [37]. They find that the Babinski type of plantar reflex is elicited by the same type of stimulus as reflex flexion of the limb, and they regard it as the minimal motor reflex response of the limb. Babinski, on the other hand, says that the pathological plantar response and the "defence reflexes" are separate phenomena [8].

Whether this be so or not, there can be no doubt that the work of Sherrington [36] throws a new light on the phenomena of spastic paralysis. In his studies on the physiology of the nervous system, he has used decerebrate and spinal animals. These differ in that the former shows a maintained tonus of the extensor muscles of the limbs, that is, of the muscles maintaining the animal in the erect posture, while the spinal animal has not this tonus. Among the reflex movements found in these animals are some of protective nature and others subserving the function of locomotion, and the fact of such great significance for the study of spastic paralysis is that Sherrington has revealed the existence of two reflex systems in the brain-stem and in the cord, each governing its own group of muscles and giving rise to reflex reactions peculiar to itself.

One reflex system employs the extensor muscles of the limbs. These are engaged in a *static* reflex; that is, the reflex which they subserve determines a condition of *tonus* in them. This Sherrington regards as the expression of posture [38]. The centre governing this reflex has been shown to be higher than the cord, in the brain-stem, and section of the brain below the level of the pons abolishes this reflex tone.

The other reflex system employs the limb flexors. The reflex reactions of this system are characteristically *phasic*, or clonic, in nature. They give rise to reflex *movements*. In the decerebrate animal both systems are in activity, in the spinal animal the static reflex of posture is absent and we see the phasic, spinal reflex system alone active. We have, therefore, two physiological groups of muscles, each engaged in a type of reflex reaction peculiar to itself. The nervous mechanisms of these are differently represented in the nervous system, the tonic extensor system requiring the presence of a "prespinal" centre, the phasic flexor system being purely spinal. One of the most striking reflexes described by Sherrington is the flexion reflex of the hind limb [37]. This consists of a single flexion, or withdrawal, of the limb in response to nocuous stimuli applied to any part of the limb surface, to any of its deep structures, or to any afferent nerve of the limb. This reflex flexion is constant except in the case of stimuli applied to the attached base of the limb, where reflex extension is often evoked. The flexion reflex is at times accompanied by synchronous extension of the contralateral limb. It is protective in function. "Spinal stepping" is also obtained from such animal preparations, but by a totally different type of stimulus.

Surely these physiological facts must have some value in the elucidation of pathological conditions in man. Sherrington [36] says: "Just as certain agents" (strychnine and tetanus toxin) "display their action more obviously in one member of these paired systems than in the other, so processes of disease may be expected to deal with the two systems unequally, and to reveal more obviously and affect more deeply one of them than the other."

It is here that we must pay tribute to the genius of Hughlings Jackson [25]. In speaking of the rigidity in hemiplegia, he says: "My speculation is that the rigidity is owing to unantagonized influence of the cerebellum. I believe that in health the whole of the muscles of the body are doubly innervated, both by the cerebrum and the cerebellum, there being a co-operation of antagonism betwixt the two great centres. Whilst the cerebrum innervates the muscles in the order of their action from the most voluntary movements (limbs) to the most automatic (trunk), the cerebellum innervates them in the opposite order. This is also supposed to be the order of their degree of influence on parts, and is equivalent to saying that the cerebellum is the centre for continuous movements and the cerebrum for changing movements."

"The muscles which, on the above suggestions, are chiefly innervated

by the cerebellum are those which have the fewest different, the most tonic, the most confluent movements, the reverse for those which the cerebrum chiefly innervates. Thus, in walking the cerebellum preserves the equilibrium of the body, tends to stiffen all the muscles; the changing movements of walking are the result of cerebral discharges overcoming in a particular and orderly way the otherwise continuous cerebellar influence. This is what is meant by a co-operation of antagonism." This quotation at length demands no apology, so significant is this hypothesis in view of Sherrington's work. The existence of a double system of motor innervation, so ably foreshadowed by Jackson, has actually been demonstrated in animals. That recent work has shown that the cerebellum itself is not the seat of the functions which Jackson assigned to it, is of minor importance; the significant fact is that he believed in the existence of two systems of motor innervation, each acting on its own group of muscles in a manner peculiar to itself and giving rise to reactions of two different types, tonic and clonic. Moreover, he recognized that disease could affect these systems separately.

Chapter II.—Methods of Investigation.

In man it is not possible to analyse reflex movements with the detailed accuracy possible in animal experiments, in which Sherrington [37] has been able to isolate and expose individual muscles, thus ascertaining the part each plays in a given reflex. We have to be content with the method of inspection to reveal reflex movement, and in the case of reflex muscular contraction without movement, palpation is the only method available. However, by means of graphic methods it has been found possible to obtain permanent records of certain reflex movements, in which the time relations and the form of the movements are seen (see Appendix II).

The clinical examination of the reflexes necessitates a study of the following points; the attitude of the limb as the patient lies in the dorsal position in bed, the voluntary power, the muscle-tone and the relative distribution of these in the different muscle groups, and the type of contracture present.

The condition of the tendon-jerks also demands attention, and in addition to the ordinary method of eliciting the knee-jerk, a quadriceps-jerk may be sought by percussion over the heads of the metatarsals

on the sole. This area has been described by Cohn and Loewy [28] as the "reflexogenous plantar zone of quadriceps." According to these authors, it yields on percussion a quadriceps-jerk in conditions of increased tonus, but I shall show that the response varies according to the type of stimulus and according to the facility of the reflex flexion in the limb. The knee- and ankle-jerks are regarded in this paper as a measure of the state of tone in the *extensor* muscles of the limb, and in these alone.

The hamstring-jerks obtained by percussion of the tendons of semitendinosus, semimembranosus and biceps femoris are not mentioned in the extensive literature devoted to the subject. They are best elicited by laying the index finger across the tendons at their insertions and then percussing the finger. In this way the muscular contraction is readily felt when direct observation is prevented by obesity and other causes. There can be no doubt that the muscular contractions obtained in this way are true tendon-jerks and are not due to direct mechanical excitation of the muscle-fibres, since in tabes, in which mechanical excitability is preserved but the tendon-jerks are abolished, these hamstring phenomena are absent too. They afford evidence of the state of tone in the limb *flexors*.

In observations on the reflex movements it is essential to record the site and nature of the stimulus evoking the reflex and the characters of the reflex motor response; that is, the muscles which contract and the order in which their contractions appear. The influence of initial posture on the form of the reflex movement and the presence or absence of reflex movement in the opposite limb also require attention, and finally the relation between the type of reflex present and the distribution of tone and power in the muscles of the affected limb is of the utmost significance.

The detailed study in the investigation on which this paper is based of a large series of cases of spastic paralysis of all kinds, sixty cases of spinal lesion and twenty of cerebral lesion, has given certain constant findings, which justify very definite conclusions as to the nature and cause of the reflex phenomena of the lower limbs. These present the closest possible analogies with the reflexes described by Sherrington in animals under experimental conditions, and there is, as will be shown, strong evidence of the existence in man of the two reflex systems found in the brain-stem and in the cord of animals.

Chapter III.—The Flexion Reflex.

The most frequent and constant reflex movement occurring in the lower limb is that of flexion. I shall speak of this as the flexion reflex. It can be obtained by a wide range of stimuli, cutaneous and deep. The movement takes place at all joints and is very constant in form, varying in amplitude alone according to the conditions of stimulus. It is also seen in the spontaneous flexor spasms of many cases of spastic paraplegia.

A complete flexion reflex consists of a single flexion at hip and knee with dorsiflexion of foot and toes, and the flexion is steadily maintained during the application of the stimulus and is not alternating in character.

Direct examination reveals contraction in the following muscles in this reflex; tensor fasciæ femoris, rectus femoris, semitendinosus, semimembranosus, biceps femoris, adductor longus, extensor longus hallucis, tibialis anticus, and extensor longus digitorum. In certain conditions there is also contraction of the peronei, but this is not an essential part of the reflex response. Probably other muscles are involved, e.g., iliopsoas and pectineus, but under clinical conditions complete analysis of reflex movement is not possible, since we are limited to inspection and palpation as means of investigation.

When the reflex is fully developed, whatever be the intensity of the motor response, these muscles all contract, even though the contraction may not be sufficient to cause movement. As is the case with the flexion reflex of the spinal animal, a strong reflex differs from a weak one, not in the number of muscles involved, but in the force of contraction in them. In certain conditions, however, such as "spinal shock," this is not strictly true, and the threshold of stimulation of the different muscles does vary to a limited extent.

The flexion may not, in certain cases to be considered later, be maintained at a uniformly high level and then gives the appearance of alternating flexion and extension, but no true alternation occurs and the flexion reflex of the limb is a single flexion of the limb at all joints varying in duration with the duration of the stimulus (see p. 300).

This reflex flexion of the limb I shall speak of as *the flexion reflex of the lower limb*, and I now proceed to study it in detail. Certain other reflexes, however, also occur, such as crossed extension and homolateral extension; these will be dealt with in turn.

It may be pointed out that homolateral reflex extension is an

unusual phenomenon, and that, as a general rule, admitting of but rare exceptions, sensory stimulation of a spastic leg evokes reflex flexion as "end effect."

§ 1.—*The Receptive Field of the Reflex.*

Sherrington has named the total area from which a reflex may be elicited, the receptive field for the reflex.[1] In the reflex under consideration in man this is very extensive, and when typical includes the skin and deep structures of almost the entire limb. The reflex is most readily obtained from the sole, the distal extremity of the limb, and here the threshold of stimulation is lowest. When reflex activity is considerably lessened, as after a complete transverse lesion of the cord, or in the coma preceding death, the field is confined to the sole and no reflex response can be obtained from any other part of the limb.

When the reflex is well developed, cutaneous stimuli applied to any part of the limb surface, and even to the lower part of the abdominal wall, will elicit a reflex complete in form. However, as we recede from the free extremity of the limb, the threshold of stimulation rises, and stronger and more prolonged stimuli are needed to evoke any response.

In certain rare cases stimulation of the skin of the upper fourth of the thigh and of the perineum gives reflex extension as end effect. When this occurs I have sometimes found that there is an intermediate zone of skin where stimulation gives either extension or flexion, or extension followed by flexion, and figs. 2, 3 and 4 are from such a case.

Since also, as will be shown, the reflex can be obtained by stimulation of deep structures, the field includes the whole structure of the limb up to a certain level. In an average reflex the field extends up to the upper fourth of the thigh, but it may extend to the abdomen, or be limited to the sole.

When any crossed reflex accompanies it, the field of this is the same as that of the homolateral flexion reflex.

Within the area of the receptive field reflex flexion is invariably obtained on adequate stimulation, but certain minor differences in this flexion are seen according to the site of the stimulus. These differences in the motor response may be described as the "local sign" of the reflex in question, and they consist in a relative predominance of the contraction in certain of the muscles involved.

I have mentioned that in conditions of "shock" of the central

[1] In describing the reflex phenomena here dealt with I use wherever applicable the terminology introduced by Sherrington in his studies on reflex action in animals.

nervous system, as in cases of acute or complete transverse myelitis, or in the coma preceding death, and also in certain cases of hemiplegia, the area from which the reflex can be elicited is restricted to the sole.

The following case of acute transverse myelitis at the ninth dorsal segmental level illustrates this :—

M. W.—A complete paraplegia of sudden onset and of twelve months' duration. There was absolute motor and sensory loss below the umbilicus, with general wasting and bed sores. The lower limbs were flaccid and all the tendon-jerks absent. According to the theory of Bastian, the condition was one of complete physiological transverse lesion of the cord.

Light stroking of the sole was without effect, but heavy stroking with the end of a penholder gave a typical "extensor response" with just perceptible flexion at hip and knee. Pin-prick gave a brisk reflex response from the sole and the outer edge of the foot. Tarsus compression gave a very feeble response. No form of stimulus applied at a higher level gave any reflex response.

The same state of affairs is found in cases of spastic paralysis immediately before death. As the reflex activity of the limb lessens, the reflexogenous area becomes smaller, but until an hour or so before death a reflex response is obtainable from the sole. *We may regard the sole, therefore, as the most essential part of the receptive field.* The importance of this will be apparent when we discuss the functional significance of the flexion reflex.

Babinski [1], however, first drew attention to the fact with which all neurologists are familiar, that stimulation of the inner border of the sole often gives a normal plantar reflex—a flexor response—while if the outer border of the sole be stimulated the extensor response is seen.

This would seem to negative the view here advanced as to the extent and character of the receptive field. My observations have led me to regard this contradiction as apparent only.

It has to be remembered that the area of skin from which the plantarflexion of the hallux is obtained in these cases overlies the abductor hallucis muscle and that the plantar fascia here is thin. A strong mechanical stimulus here produces plantarflexion of the hallux by direct stimulation of this muscle, the contraction of which flexes the hallux.

Simple localized pressure, as with the end of a penholder, over this muscle will give a plantarflexion of the great toe, while applied elsewhere to the sole it gives no response. As remarked in another

part of the paper, *localized* pressure of this type does not commonly elicit any reflex response, and it seems therefore highly probable that in many cases this toe phenomenon is not a true reflex, but purely local.

In other cases where this explanation has not been found to apply, the phenomenon seems always due to the fact that the field is restricted to the outer fraction of the sole. This is often the case in hemiplegia, where this double phenomenon is most common.

§ 2.—*The Nature of the Stimulus.*

Light touch, such as the contact of cotton-wool, or of a camel's-hair brush, except in very rare instances gives no reflex response. I have only seen it thus obtained twice in the whole series of cases examined, and in these the reflex was very facile.

Light pressure to the sole, applied, not as a contact but as a stroking movement, gives in typical cases a good reflex response. This is the method in common use in eliciting the plantar reflex, and if the stimulus be gentle a dorsiflexion of the hallux may be the only visible effect, but this is never the only muscular contraction, and palpation of the hamstring tendons reveals, even to minimal stimuli, contraction of these muscles.

This type of stimulus is commonly effective in evoking a reflex response when applied to the sole and the outer half of the dorsum of the foot as high as the lower end of the external malleolus. When very facile the reflex is elicited by each stroke to the skin over this area. The threshold is lowest on the outer border of the sole and at the base of the hallux. At higher levels on the limb cutaneous stimuli of this type are commonly ineffectual.

Noxious stimuli, that is, stimuli normally painful, such as pin-prick or pinching of the skin, give a good reflex over the whole receptive field, the upper limits of which indeed are defined by the use of such stimuli. Above the level of the foot, however, repeated stimuli of whatever form are often needed to elicit a response, and they have to be correspondingly stronger the further they are removed from the extremity of the limb. Usually on the sole and dorsum a single pin-prick gives quick ample flexion, while on the thigh several stimuli delivered in quick succession may be necessary.

Stimulation of the deep structures of the limb gives a reflex identical with that obtained by the methods mentioned above. Applied to

muscles and tendons we find, as in the case of cutaneous stimuli, that stimulation of distally placed structures gives a readier response than that of proximal muscles, &c. When the receptive field is limited we find that while, for example, pinching of the tendo Achillis gives a flexion reflex, Oppenheim's procedure may be ineffectual, or that when this latter gives a reflex, pressure of the thigh muscles may be inadequate. I have only twice been able to satisfy myself that Oppenheim's procedure will elicit a reflex when all forms of equally intense cutaneous stimulation to the foot fail to do so.

Forcible compression of the tarsus and forced flexion of the toes, as practised by the French observers, are examples of stimulation of joints and muscles which give reflex flexion of the limb.

Electrical stimuli, such as the faradic current, or thermal stimuli, are equally effective in giving a reflex movement when applied to the receptive field.

The reflex contraction of quadriceps extensor femoris mentioned earlier (Loewy [28]) is an instance of a single form of stimulus, namely, a sudden localized light pressure, giving a specific motor response from a certain skin area. I have found in cases of spastic paraplegia, that where a light tap on the sole gives this movement, a heavier tap gives reflex knee extension followed by a typical complete flexion reflex; a harmless stimulus gives reflex extension, a more intense, nocuous stimulus gives also reflex flexion—or this alone.

Sherrington [36] describes a similar phenomenon in the spinal dog, in the "extensor thrust" reflex, where light pressure between the toe pads gives a reflex extensor thrust of the limb, but all other forms of stimulation, thermal, painful, or electrical, give a flexion reflex.

In cases of paraplegia with contracture in flexion where involuntary flexor spasms occur, the lightest stimulation applied to any part of the limb, even contact with moving bedclothes, gives reflex flexion; and it is in such cases alone that passive movement is an adequate form of stimulus. In these cases, any attempt to straighten the partially flexed limb provokes strong reflex flexion at hip and knee with dorsiflexion of foot and hallux.

In limbs only moderately spastic, or spastic in extension, passive movement alone gives no reflex response.

Many patients were supported astride a horizontal bar with the legs hanging free. The passively flexed limb was suddenly released and allowed to fall into extension, but no reflex flexion, or any form of reflex whatever, was to be obtained.

In conclusion, when the reflex is present it can be elicited by stimulation of various forms applied to any part of the limb, except part of its proximal extremity. Although light and painless stimuli applied to the free extremity of the limb give the reflex, yet it is most characteristically obtained by stimuli which are harmful in character, or noxious. The two types of reflex obtained from the sole by percussion exemplify the importance of the nature of stimulus in determining reflex response, but with this exception any stimulation when applied within the receptive field gives reflex flexion as its motor effect. The threshold is lowest at the distal extremity of the limb and here alone light stimuli are effective.

§ 3.—*The Motor Reflex Response. The "Extensor Plantar Response."*

I have said above that all the flexors of the limb are activated in the flexion reflex. These include hip and knee flexors and the dorsiflexors of the ankle and digits. Now, of these, the dorsiflexors, in anatomical nomenclature, are named extensors. There can be no doubt, however, that these muscles must be grouped physiologically with the flexors.

In involuntary flexor spasms and in the flexion reflex elicited as already described these muscles act with the flexors of hip and knee, while with reflex extension we always see plantarflexion of foot and digits. Of the general shortening of the limb, which occurs in the flexion reflex, dorsiflexion is the natural accompaniment.

Moreover, the lower limb of man can be compared, segment for segment, with the hind limb of the dog, and Sherrington has shown that here the dorsiflexors act with the flexors and must be regarded as such, while plantarflexion and extension at hip and knee are, on the other hand, similarly allied.

The following comparative diagram (fig. 1) shows this: A is the hind leg of the dog, and B the lower limb of man. A is after Sherrington [37].

The flexors therefore are: Ilio psoas, rectus femoris, sartorius, gracilis, pectineus, adductor longus and brevis, tensor fasciæ femoris, semitendinosus, semimembranosus, biceps femoris, tibialis anticus, extensor longus digitorum, extensor longus hallucis. These are not all represented in the diagram.

With the methods at our disposal in clinical investigation, it is not easy completely to analyse any complex reflex movement. For example, slight contraction of sartorius or rectus femoris may easily

pass unobserved. On the other hand, the slightest twitch of tensor fasciæ femoris is at once visible, while palpation of their tendons reveals the slightest contraction in the hamstring muscles.

Certain muscular contractions, therefore, are not seen. In the case of the toes, however, where the effect of gravity is negligible, as it cannot be in the case of more proximal joints, a hallux movement betrays the least contraction of the extensor longus hallux. In a feeble

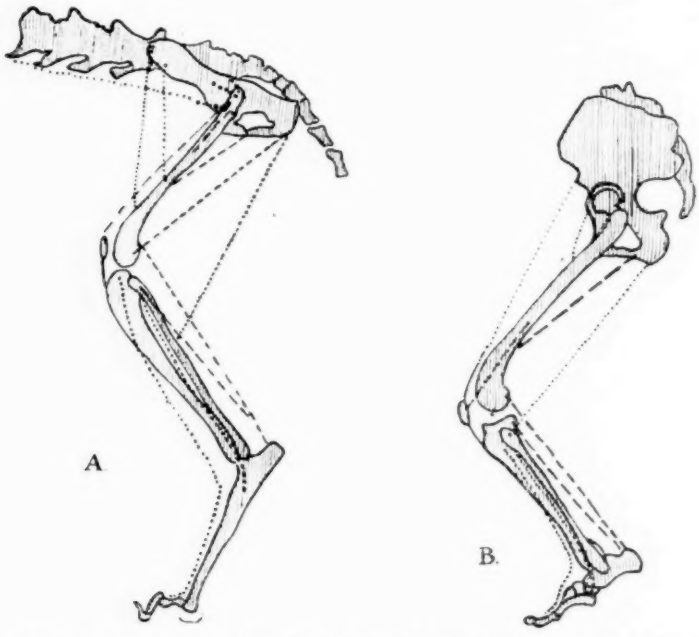

FIG. 1.—In A, the muscles activated in the flexion reflex of the hind limb of the spinal animal are indicated by dotted lines; their antagonists by interrupted lines. In B, the muscles acting in the flexion reflex are similarly indicated.

reflex this may be the only *visible* effect, but it is by no means the only one present, and careful investigation has drawn me to the conclusion *that an isolated "extensor response" does not occur.* It is invariably accompanied by contraction of the proximal flexors of the limb, and in all cases a palpable contraction of semitendinosus and semimembranosus can be felt, while in a majority where the reflex is well developed an actual movement of flexion at hip and knee obtains. The importance

of this fact in any attempt at analysis of the nature of the "extensor" type of plantar response will be at once apparent.

We know also that the flexion reflex is most readily elicited from the sole and that here light stimuli, such as are adopted to elicit a plantar reflex, suffice to give it. It becomes evident, therefore, that the "extensor response" has, in common with the flexion reflex, its site and mode of stimulation and its form, and there is no doubt that this well recognized and important sign of pyramidal lesion is part of the flexion reflex. It may be said at once that Babinski [8] refuses to accept such an interpretation, which Marie and Foix [30] have already put forward in a somewhat qualified and different form.

It might be thought, as these authors say, that the "extensor response" is the minimal response or "motor focus" of the reflex. This is not always a point easy of solution, since, as I have pointed out, the contractions of certain proximal flexors are not easy to observe and do not lend themselves to graphic recording as readily as toe movement.

A study, however, of the reflex in conditions of "spinal shock" shows that the inner hamstrings are the muscles which are earliest involved and form the "motor focus" of the reflex.

From this point of view, observations were made on three patients with hemiplegia from cerebral tumour, when comatose and at the point of death. The affected limbs became absolutely flaccid, and the tendon jerks disappeared while a flexion reflex remained still readily obtainable from a fairly wide receptive field on intense stimulation, such as pin-prick or pinching. Gradually the field became smaller until from the sole alone could a reflex be elicited. In all three instances the following phenomenon was then seen: The reflex motor response became less ample and forcible until only a toe movement was seen, but a palpable hamstring contraction remained, and in one of the three, an emaciated patient, this indeed could be seen. The field of musculature involved then shrank until the toe movement went, and the only evidence of reflex response on stroking the sole was a readily felt hamstring contraction. Finally, this went and the limb retained no sign of reflex action.

I think it justified, therefore, to regard these muscles, in man as in animals according to Sherrington, as the motor focus of the reflex. It must be admitted, though, that certain deep-lying proximal flexors could not be investigated.

Case 1 (in the Appendix) shows this phenomenon inversely. It was a case of acute complete transverse myelitis. When seen a week after

onset there was absolute flaccid paralysis with retention of urine, complete sensory loss over the lower limbs and absent tendon-jerks; judged by Bastian's criteria, a total tranverse lesion.

Heavy stroking of the sole, and here only, gave reflex flexion at hip and knee, feeble but definite. There was no foot or toe movement. The minimal motor response was hamstring contraction alone.

Within the next few days the reflex response increased, dorsiflexion of foot and finally of hallux appearing. From this time on an "extensor response" could be obtained, but hamstring contraction still remained the minimal motor response to slight sole stimulation.

Here we can see the variation in threshold of contraction of the different muscles, a variation which is not evident in the fully developed reflex. The muscles disappear from the reflex response in the inverse order to that in which their contractions first appear.

The "extensor plantar response" is therefore not the minimal motor response, and it is never obtained without contraction probably of all the limb flexors, certainly of some proximal flexors. *It is not separable from the flexion reflex.*

Is limb flexion ever associated with plantarflexion of foot and toes, i.e., with a so-called "flexor response"? This question brings us to the consideration of "local sign" in reflex response.

It has been shown that within the limits of the receptive field an adequate stimulus gives only one type of reflex response, namely flexion of the limb. Yet it was long ago pointed out by Babinski [1] that the pathological type of plantar reflex varies somewhat in form according to the site stimulated. He observed that stimulation of the outer border of the sole gave eversion of the foot as well as the toe movement, while from the inner border of the sole inversion of the foot occurred. Further he says that, in some instances, limb flexion when elicited from the thigh is accompanied by plantarflexion of the toes—a "flexor response" [8].

It is found that when the flexion reflex is present, dorsiflexion of the hallux always results from stimulation of the sole, and is accompanied by contraction, more or less forcible, of the limb flexors. Stimulation of the upper limits of the field gives the same type of reflex, but certain modifications are sometimes present. These are that the contraction of the distal muscles is less powerful, and there may be little, if any, "extensor response" in such cases, which, however, are the exception. In two of the instances in which I have observed slight actual plantarflexion accompanying limb flexion, this was not an active

movement but a passive pull when the foot dorsiflexed, since if the dorsiflexion of the foot was prevented and the angle between foot and leg maintained constant, this movement did not occur.

In another class of case active plantarflexion does occur (*see* figs. 2 and 3), but graphic records show that it precedes a dorsiflexion movement which synchronizes with the limb flexion.

In a case of this type, which was one of syphilitic focal myelitis with spasticity in extension, exaggerated tendon-jerks, clonus and Babinski type of plantar reflex, pinching of the skin of the thigh gave the

FIG. 2.—Records a series of hallux movements obtained by stimulation of the skin of the limb. An upward movement is one of dorsiflexion, and a downward excursion represents plantarflexion. Six stimuli were applied in rhythmic succession; the first and last to the sole, the others consisted of pinching the skin on the inner surface of the thigh. Sole stimulation gave a pure "extensor response." Of the thigh stimuli, the first gave simple dorsiflexion. The others gave always plantarflexion, a "flexor response," followed by an "extensor response" (time in seconds).

following results. The first stimulus gave invariably a pure flexion reflex with an "extensor" plantar response. Subsequent stimuli, however, in a rhythmic series, gave a plantarflexion of toe and foot followed by limb flexion including foot and toe dorsiflexion (fig. 2). The whole movement was so brisk as to leave the impression that the reflex movement consisted of a "flexor response" and limb flexion. The records (figs. 3 and 4) prove that this is not so and demonstrate the identity of the "extensor" type of reflex response with the flexion reflex.

In this case, stimulation of the skin of the perineum gave reflex extension of the limb with plantarflexion of foot and toe, and there was,

therefore, a transitional zone from which, on stimulation, there was obtained a reflex extension followed by a reflex flexion.

A third class of case, in which this anomaly may appear, is in old-standing paraplegias with contracture of the toes in plantarflexion. In such a case a dorsiflexion of toes is impossible. It would be superfluous to emphasize this were it not that such a case has been used by Babinski [8] to prove that these two phenomena of limb flexion are separable and different in nature.

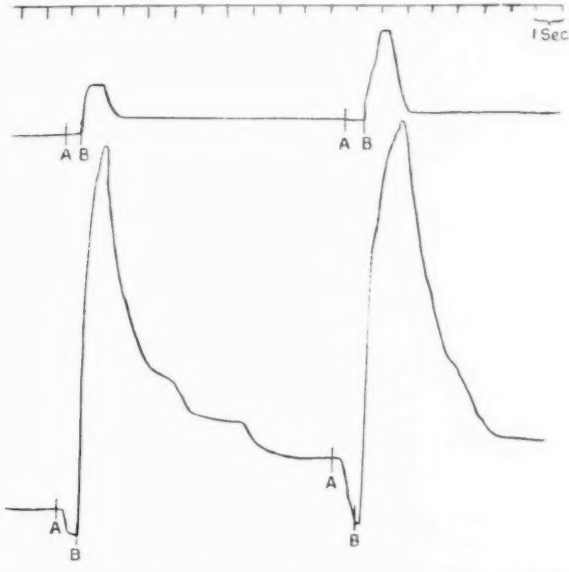

FIG. 3.—Simultaneous comparative records of toe movement and limb flexion at hip and knee. The upper tracing records only limb flexion, and was taken from the external malleolus. The lower, taken from the tip of the hallux, records the plantar reflex also. The vertical strokes (A and B) through the records indicates corresponding places in the two cases. The stimulation was in each instance a pinch of the skin on the inner side of the thigh (upward movement = dorsiflexion). These show that the "flexor response" consists of a feeble plantarflexion of hallux, which precedes the ample dorsiflexion of foot and toe, this latter being synchronous with the limb flexion of which it is a part.

The consideration of these cases in which plantarflexion of foot and toes is associated with limb flexion, raises the question of the relationship between the normal "flexor response" and limb flexion as a whole. I shall later refer to some of the characteristics of the normal plantar response in detailing the differences which exist between it and the

crossed plantar reflex. The normal plantar reflex, as I shall there describe it, consists of plantarflexion of digits, and is accompanied by dorsiflexion of the foot, contraction of tensor fasciæ femoris, and certain other variable contractions of the limb musculature.

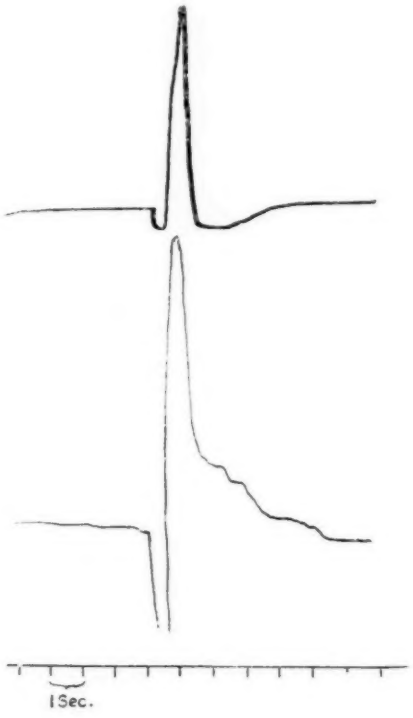

FIG. 4.—Synchronous records of hallux and foot movement in a diphasic response, such as those in figs. 2 and 3. The upper was taken from the ball of the foot by means of a thread connected with a writing lever. The lower tracing was similarly taken from the tip of the hallux. They show that in the preliminary plantarflexion, both toe and foot take part, just as in the subsequent "extensor response." In each case the hallux movement is the more ample.

In some cases, apparently flexor and extensor movements are combined in varying proportions, so that, as far as my investigations go, the normal "flexor response" does not appear to form part of any systematic reflex movement of flexion, such as seems to be the case with the "extensor response." It is open to question whether the normal plantar

reflex is a co-ordinated movement involving contraction and relaxation of opposing muscle groups, that is, "reciprocal innervation," such as the pathological reflex undoubtedly is.

§ 4.—*Involuntary Flexor Spasms and Paraplegia in Flexion.*

In this description of the reflex movement, I have so far dealt with a definite reflex elicited by a definite, known stimulus. What place do the involuntary flexor spasms, so prominent in some cases of spastic paraplegia, take?

Examination reveals that each flexor spasm is a powerful flexion reflex complete in form. These spasms occur most frequently in cases of paraplegia in flexion, and as the extended type of paraplegia passes on to this severer form, the flexor spasms increase in strength and frequency. Until Babinski drew attention to the flexed form of spastic paraplegia and showed that it possessed constant characteristics, it was the custom, and still is in this country, to dismiss it lightly as differing from the form in which the legs are extended, only by the presence of contracture in the flexors. But, as Babinski [5] has pointed out, this so-called "contracture" is an active muscular contraction, and permanent fibrous contracture is but a terminal phenomenon. Moreover, the condition is characterized by complete intermissions of the spasm, which result in the production of alternating flexion and extension movements. Of these the former are active, the latter generally passive and due to relaxation of the flexor spasm. Legs spastic in extension show no such variations.

In paraplegia in flexion we have a condition in which the extensor muscles show little or no trace of reflex action, and in which the flexors are, therefore, unantagonized. The intermissions described by Babinski are periods between successive flexor spasms, or flexion reflexes.

Even when this condition of complete absence of reflex action in the extensors has ensued in a progressive paraplegia, the reflex activity of the flexors remains characteristically phasic, and a permanent attitude of the limb in flexion only appears when actual contracture has set in in the muscles. Palpation of the tendons of the hamstrings reveals the markedly intermittent nature of the spasms, and it is when these are minimal or absent that hamstring-jerks are to be obtained most readily.

Whether these flexor spasms are actually spontaneous or not is uncertain. Any manipulation of the limb, or any light cutaneous stimulus such as that of cold when the bedclothes are removed, increases the spasm. Also since we know that an intact afferent path from

muscles to cord is essential (Sherrington, Foerster) it seems probable that we are only dealing with a condition in which the threshold of stimulation is extremely low.

Flexor spasms also occur in paraplegia in extension, and in a lesser degree in hemiplegia. In the former case they are usually alternating in time in the two limbs, not simultaneous. In paraplegia in flexion they are often bilateral and synchronous.

The involuntary spasm typical of the extended form of spastic paralysis, however, consists of a sudden increase of extensor tone, usually in both limbs at once, and not producing any actual movement. This jerky spasm is quite unlike the slow powerful flexion reflex, and is allied to the spontaneous limb-clonus so often seen in these cases.

§ 5.—*Reciprocal Innervation in the Flexion Reflex.*

So far I have dealt solely with the muscular contractions in the flexion reflex. Every co-ordinated movement involves, as Sherrington has shown, both excitation and inhibition; excitation and contraction of the "prime movers" (Beevor), the flexors in this case, with inhibition and relaxation of their antagonists, the extensors.

Certain muscles are pure flexors, e.g., sartorius; others act as flexors of one joint and extensors of another. Such muscles are the hamstrings, which extend the hip and flex the knee. In the flexion reflex these muscles act as flexors, their extensor action being completely overcome by the hip flexors. Rectus femoris also illustrates this and the further point that muscles which are regarded as units anatomically are not so physiologically; thus, in the flexion reflex, rectus acts as a flexor, while the vasti, being pure extensors, are inhibited. Yet these form part of one muscle—quadriceps extensor femoris. Beevor has drawn attention to this separate action of rectus femoris [10].

In the case of pure extensors, such as the vasti, we have clear evidence of their inhibition during the progress of a flexion reflex. The knee-jerk and knee-clonus are evidence of reflex activity in the extensors, and these phenomena are diminished or even abolished by a flexion reflex.

It has long been known that a sudden pinch of the skin of the limb would stop an ankle-clonus, and Brown-Séquard [11] pointed out that clonus ("spinal epilepsy") of a spastic limb could be stopped immediately, and the limb rendered "perfectly supple and pliable" by forcible plantar-flexion of the hallux, but no definite explanation has ever been forthcoming. In the light of the present investigation the cause is clear.

It is found that if the knee-jerk in a case of spastic paralysis be rhythmically elicited and the sole of the limb be then briskly stroked for some seconds, the jerk commonly diminishes in force, even when the reflex flexion is not sufficient to prevent it mechanically.

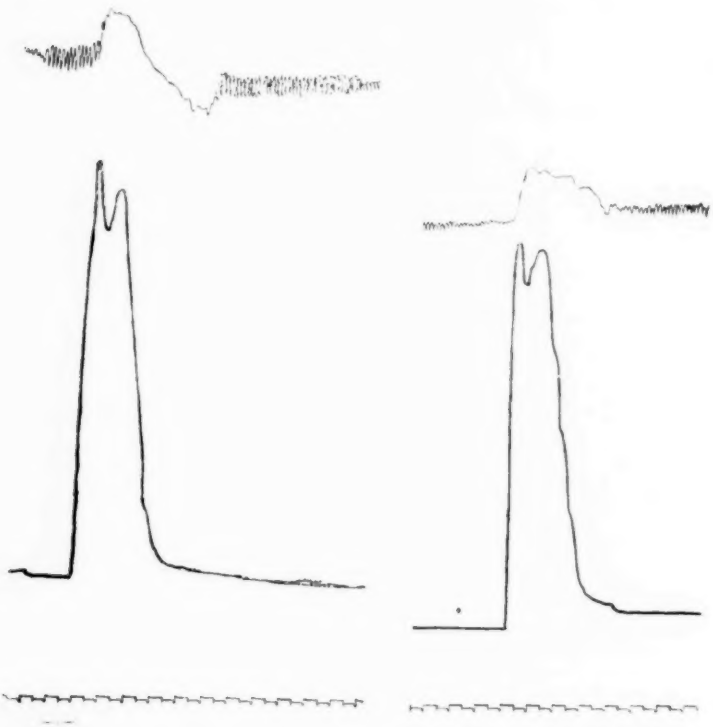

FIG. 5.—The upper tracings record a spontaneous knee-clonus, the lower an "extensor response," elicited by stroking the sole sharply. There was no power of voluntary flexion of the limb, which was fully extended when recording the reflex movements. While active flexion was in progress the clonus invariably ceased, reappearing at once on the cessation of the flexion reflex. The upward excursion in the upper record was due to some flexion at hip and knee accompanying the "extensor response," and does not represent any active movement of the patella with which the lever was connected.

The phenomenon can be recorded graphically in the case of a spontaneous knee-clonus which can often be inhibited by light sole stimulation. Fig. 5 is such a record taken from a case of extra-medullary spinal tumour of 7th cervical level, causing spastic paresis

of the lower extremities in extension. The extensor rigidity was intense and spontaneous knee-clonus was frequent. All the tendon-jerks were increased, and the plantar response was "extensor" in type.

Light sole stimulation gave a flexion reflex with very little actual limb flexion, but an ample toe movement. While this lasted the knee-clonus ceased, and recurred when the flexors ceased to be actively contracted; a beautiful example of reciprocal innervation in the flexion reflex.

In fig. 5 note that the upward movement in the clonus curve is due to limb flexion—not to vastus contraction and traction on the patella. This flexion movement could not be abolished completely. The crossed knee-clonus is affected in quite a different manner and will be dealt with in the next section.

We have evidence, therefore, that extensor tonus is inhibited by active contraction of the flexors in the flexion reflex, and we shall discuss, at a later stage, the effect of this constantly maintained extensor tonus on the flexion reflex.

§ 6.—*Reflexes accessory to the Flexion Reflex: Crossed and Homolateral Extension: The Crossed Plantar Reflex.*

Reflexes accessory to the flexion reflex are the crossed extension reflex, the crossed plantar response, and the homolateral reflex extension obtained, in some cases, by stimulation of the skin of the base of the thigh, or of the perineum. This last is independent of the flexion reflex, although it occasionally seems to occur in association with it. Reflex extension is commonly seen, however, in association with reflex flexion as a crossed phenomenon.

Babinski [1], Byrom Bramwell [12], Fairbanks [19] and others have recorded that in certain cases of spastic paralysis in which an "extensor" type of plantar reflex is obtainable, there occurs, on stimulation of the crossed foot, a plantar reflex of "flexor" type. Thus in hemiplegia an "extensor" plantar reflex is obtained from the affected limb, but on eliciting a plantar on the sound side a "flexor" plantar response is seen on the affected side.

Examination of this crossed response shows that it differs fundamentally from the normal plantar reflex, the so-called "flexor response."

In this we see plantarflexion of digits, dorsiflexion of foot, a constant contraction of tensor fasciæ femoris and certain inconstant contractions of the limb musculature.

A

B

FIG. 6.—Homolateral and Crossed Plantar Responses. A, resting position of limbs; B, plantar stimulation of left sole giving flexion reflex (flexion at hip and knee was forcibly prevented by an assistant). On right side a "crossed plantar response."

The crossed plantar response, examined in the extended limb, consists of plantarflexion of both foot and toes, but no tensor fasciæ femoris contraction.

The contrast is well seen on eliciting a plantar reflex from the normal sole of a hemiplegic in whom the crossed reflex occurs. The dissimilarity between the two is striking.

This crossed reflex would, therefore, seem to be indicative of active extension of the limb, and this is readily capable of proof. If the limb

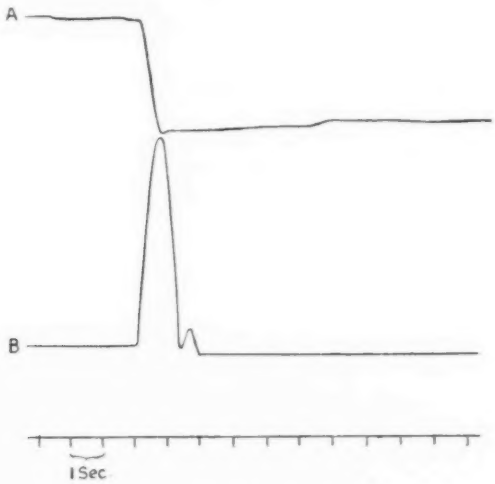

FIG. 7.—Flexion and Crossed Extension Reflexes. A, left limb. This was held supported in flexion by an assistant—one hand under heel, and one over knee to prevent the limb falling laterally; B, right limb. This lay extended and the sole was stimulated by a brisk stroking. Both records are one-eighth of the amplitude of the actual excursion of the limb. Flexion is an upward, extension a downward, movement.

be previously flexed and supported lightly so that no resistance is offered to active extension and the crossed sole then stimulated, there occurs an active extension at hip and knee as well as the movements already described. This is the crossed extension reflex.

The crossed "flexor response" is, therefore, part of a crossed extension reflex. Crossed extension and the crossed plantarflexion, when present, commonly occur together. Rarely the latter is seen alone or without a trace of extension at the hip and knee, and I conclude that, as in the spinal animal, crossed extension may be present only at the distal joints.

Crossed extension at hip and knee without plantarflexion of foot and toes I have not seen.

It is the general rule in cases of spastic paralysis with rigidity in extension and increased knee- and ankle-jerks for the crossed extension reflex to be present, but it is not constantly present. Two photographs and a graphic record (figs. 6 and 7) from a case of spastic paraplegia due to meningo-myelitis show these reflexes.

In this case the legs showed moderate paresis with strong extensor rigidity. All the tendon-jerks were exaggerated, and there was knee- and ankle-clonus, and "extensor responses" from the soles. Crossed extension was constant and very active. When the crossed limb, right or left, lay extended, this was seen as plantarflexion of foot and toes, but when flexed, active extension occurred at all joints.

The photographs (fig. 6, A and B) show the resting attitude of the limbs and the flexion reflex on the stimulated side with the crossed plantar response. The combination of plantarflexion of foot and toe is well seen, and it is obvious that the crossed reflex is not the normal "flexor plantar response."

Fairly strong stimulation of the sole was needed to evoke a good crossed extension and minimal stimuli gave plantarflexion of foot and toe alone, the motor focus evidently being distal in this reflex.

The crossed extension reflex is always, in my experience, a single and not an alternating reflex. There is, it is true, often a flexion rebound after active extension, which is seen as an "extensor response" with slight flexion at all joints. If the whole movement be very rapid, this crossed "extensor response" is apparently synchronous with the homolateral flexion reflex, and I think that the "crossed Babinski" which has been described is of this type, and is not synchronous with flexion in the stimulated limb, but is a rebound following crossed extension, such as Sherrington [36] describes. Figs. 8 and 9 show this photographically and graphically.

The case from which these were obtained was one of acute disseminated sclerosis. When the patient first came under observation the legs showed spastic paresis in extension with a well-marked flexion reflex and crossed extension reflex. The condition in the legs progressed and became that of paraplegia in flexion; as this happened the crossed extension disappeared. (This case is described at length in Appendix, Case 2.)

A well-marked flexion reflex may be present without any evidence of crossed extension, which is no integral part of the reflex. The factor

determining absolutely the occurrence of the crossed extension reflex is the presence of reflex activity in the extensor muscles of the limb. The indications of reflex activity of this group of muscles are increased knee- and ankle-jerks, the presence of clonus and spasticity in extension.

Fig. 8A.

Fig. 8B.

FIG. 8A.—Resting attitude of limbs.

FIG. 8B.—Flexion reflex with "crossed plantar response," consisting of plantarflexion of foot and toes.

These are the conditions found in hemiplegia and in paraplegia in extension, in which alone crossed extension is seen. It is not seen in paraplegia in flexion, which I have found to be characterized

Fig. 8c.

FIG. 8c.—"Rebound" in each limb in opposite sense to original movement.

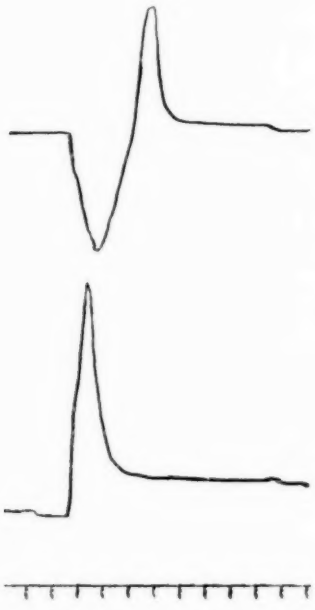

FIG. 9 is a graphic record of the phenomenon shown in fig. 8. The upper tracing shows a crossed plantarflexion followed by an "extensor response"; "rebound" phenomenon. The lower tracing, simultaneously taken, records an "extensor response" elicited from the left sole. The crossed extension is synchronous with this; the crossed "extensor response" is later. This is a direct record of hallux movement. Upward movement indicates dorsiflexion, and downward movement plantarflexion. Time, below, in seconds.

by the absence or diminution of the reflex activity of the limb extensors, while in the flexors this is conserved and increased. The stimulus and receptive field for the crossed extension reflex are those of the homolateral flexion reflex.

In this reflex the muscles which contract are those which in the flexion reflex are inhibited. This increased tone in the extensors I have been able to record graphically, in the case of the knee extensors.

In a case of spastic paraplegia of Erb's type in which there was spontaneous knee-clonus, the eliciting of a plantar reflex, i.e., a flexion reflex, caused an inhibition of the knee-clonus on the same side, but so increased the tonus of the crossed extensors that they went into well-marked tonic spasm, which relaxed and resumed the clonic form on cessation of the stimulus (fig. 10).

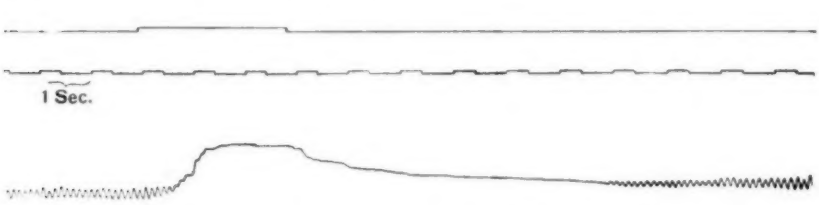

FIG. 10.—Records a spontaneous vastus clonus becoming strong tonic spasm during plantar stimulation of the crossed leg. The upper signal records the duration of the sole stimulus. The lower signal gives time in seconds (two segments to 1 sec.).

In the case from which figs. 8 and 9 were obtained, the same phenomenon was demonstrable in a different way. The condition passed from that of paraplegia in extension to paraplegia in flexion, the right leg being affected earlier than the left. The right knee-jerk failed and soon disappeared. For two weeks after the knee-jerk had ceased to be obtained by ordinary means alone, it could be caused to reappear by simultaneous stimulation of the crossed sole, and for as long as this stimulation lasted. Unfortunately, the jerk thus obtained was too feeble to lend itself to graphic record. But for a period of at least two weeks this reinforcement of the crossed extensors could be seen.

Also, evidence of inhibition of the crossed flexors during a flexion reflex is often obtainable. If, by gentle continued stimulation of one sole, in a case of spastic paraplegia, the hallux be induced to assume an attitude of dorsiflexion, similar stroking of the crossed sole will cause a definite plantarflexion of the toe originally dorsiflexed, in spite of continued stroking of the sole. Of course, increased intensity of the original

stimulus will cause the reappearance of the flexion reflex originally elicited. This brings me to a further fact that the homolateral reflex will, if sufficiently intense, always break through and overcome a crossed reflex of whatever intensity. That is, a relatively weak flexion reflex is more potent than the most intense crossed extension reflex.

The rebound following reflex flexion and crossed extension are also accessory to the flexion reflex, since they follow it. Sherrington [36] has described in all reflex reactions a "post inhibitory exaltation" of activity of the muscles inhibited during the reflex. This certainly occurs in the flexion reflex and crossed extension reflex. In the former as rebound extension, and in the latter as rebound flexion. The latter I have already spoken of, and figs. 8 and 9 illustrate it.

Homolateral extension reflex.—This is of relatively rare occurrence, and I have seen it only five times in the series of cases examined. It is obtained, as mentioned earlier (*see* p. 285), on stimulation of the skin, by pin-prick or pinching, of the upper extremity of the thigh and of the perineum, and consists in a quick extension at hip and knee with plantarflexion of ankle and toes. On the few occasions I have seen it, I have not observed any crossed reflex accompanying it.

Sherrington describes a similar reflex occurring on stimulation of the base of the limb or of the perineum by painful stimuli in animals.

§ 7.—*Factors modifying the Reflexes.*

(*a*) *Muscle Tone in the limb.*

Thus far, I have described the features of a typical flexion reflex and of a crossed extension reflex. Whenever obtained, the flexion reflex consists of a single movement of flexion of the limb, but it is quite clear that the groundwork of muscle tone into which the reflex breaks must have a modifying effect on it, while in the case of the crossed extension reflex its existence depends on the presence of hypertonus in the extensor group of muscles in the limb.

It is not my intention here to go deeply into the question of contracture and hypertonus, but, regarded broadly, the lower extremity may show either of two conditions in spastic paralysis :—

(1) It may be a limb rigid in extension, with hypertonus predominant in the extensors, which muscles show also, as signs of increased reflex activity, increased knee-jerk and ankle-jerk with clonus. The flexors also show great increase of reflex activity, e.g., the flexion

reflex with the Babinski plantar response, involuntary flexor spasms and increased hamstring-jerks. Such a condition is seen in hemiplegia.

(2) It may be a limb spastic in flexion. In such a case we find evidence that the reflex activity of the extensors is greatly diminished, or absent. The knee-jerks and ankle-jerks are feeble or unobtainable. There is no clonus. The extensors show no hypertonus. The flexors, on the other hand, show extreme reflex irritability. There is a well-marked flexion reflex with its Babinski plantar phenomenon. Involuntary flexor spasms are continual, and reflex flexion is provoked by the slightest cutaneous stimulus to the limb. The hamstring-jerks are very brisk. The muscles do not, it is true, show the steadily maintained hypertonus characteristic of the extensor rigidity, but the limb tends eventually to go into a permanent attitude of flexion. Even then, however, until contracture becomes marked, there are periodical remissions of the flexor spasm, and the legs fall into extension transiently.

In speaking above of the predominance of extensor spasticity, in cases of spastic paralysis in extension, I do not necessarily imply that when a lesion gives rise to such a condition the extensors are *more* affected than the flexors. I would rather suggest that they are *differently* affected, and that while the extensors show their separation from a higher centre, or their liberation from cortical control by entering in a condition of maintained hypertonus, which might, using Sherrington's phraseology, be called a "static reflex," the flexors indicate their liberation characteristically, in the appearance of the flexion reflex—a "phasic reflex." Both muscle groups show increased tendon-jerks.

Whether this view be accepted or not, it is certain that the leg, in cases of spastic paralysis, must conform to one or other type, except in conditions of "spinal shock," when the extensors show no sign of reflex activity and that of the flexors, though present, is minimal. These special conditions will be discussed later.

We are able then, except in this third case, to examine the flexion reflex under these two conditions; with and without the presence of extensor reflex activity in the limb.

These two types of paraplegia, the clinical characteristics of which have been emphasized by Babinski [2, 5], and named by him "paraplegia in extension," and "paraplegia in flexion," give us the opportunity of examining the effect which a groundwork of hypertonus in the extensors has on the flexion reflex. This reflex employs the flexors, while the antagonists of these muscles, the extensors, are by reciprocal innervation inhibited during the progress of the reflex.

The flexion reflex in paraplegia in extension.—In this type of spastic paralysis the flexion reflex is characterized by being less easily elicited in the ample form seen in paraplegia in flexion. The toe movement may be the only actual movement visible, though palpation of the hamstrings will reveal invariably a contraction of these muscles.

The reflex movement is, when it involves actual flexion at hip and knee, always rapid, and is followed immediately on cessation of the exciting stimulus by quick active extension "rebound." It is in these circumstances that intermittent flexion is found. Fig. 11 records this unequally maintained flexion. In this there is no true alternating flexion and extension. The condition is one in which a flexion reflex incompletely overcomes a strongly maintained hypertonus in the antagonists; a static reflex is broken into imperfectly by a phasic reflex.

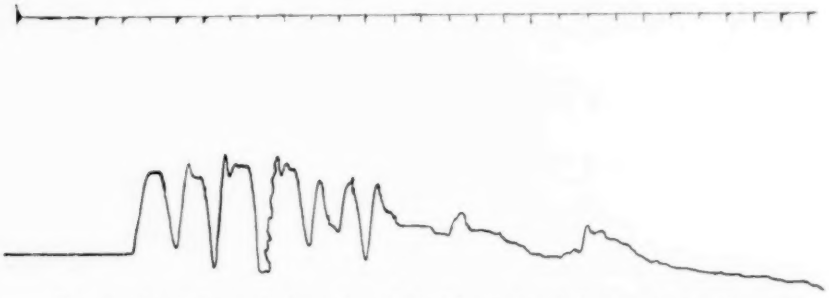

FIG. 11.—Intermittent flexion reflex obtained by continued pinching of the skin of the thigh. The stimulus is not recorded, but it did not cease until reflex movement had already died away. Signal records time in seconds.

The case from which this record was obtained was one of cerebrospinal lues, with spasticity of the legs in extension. In these circumstances the flexion reflex consisted of an excitation of the flexors, which broke into a maintained hypertonus of the extensors. This it temporarily and imperfectly inhibited and replaced.

Dependent also on this extensor tonus is the crossed extension reflex, which never occurs except when the extensors show increased reflex activity. In cases of increasing paraplegia in which the legs go into an attitude of flexion, this crossed reflex disappears *pari passu* with the diminution of extensor hypertonus, knee-jerk and ankle-jerk.

The crossed extension reflex is only seen therefore, clinically, in cases of hemiplegia and of paraplegia in extension. It does not occur in paraplegia in flexion (*see* Case 2, in Appendix).

The flexion reflex in hemiplegia is very like that obtaining in paraplegia in extension as regards extensor rebound and the presence of the accessory crossed extension reflex. In hemiplegia, however, the severance of the lower motor centres from voluntary control is less complete than in most cases of paraplegia, so that the flexion reflex tends to be more facile in the latter. There are other modifications, too. The receptive field is often confined to the foot, sole and dorsum, for cutaneous stimuli, and to below the knee for deep muscular stimuli (Oppenheim's procedure). The response is seldom ample, but is true to type in that the "Babinski response" is accompanied by palpable hamstring contraction. The reflex, in other words, though present and complete in form, is less facile and less ample; less uncontrolled.

Crossed extension is present, and can be elicited by stimulation of the sole on the normal side.

In paraplegia in flexion.—In these cases the flexion reflex reaches its greatest development, and in the transition from paraplegia in extension to this condition the involuntary flexor spasms increase in power and frequency, until an attitude of flexion is attained. Even in this the phasic character of the reflex is seen in transient intermissions, partial or complete, of the flexor "spasticity."

The receptive field is very wide and may reach the abdominal wall for pin-prick. Light stimuli suffice, and the minimal reflex responses are complete, ample and strong.

In these cases the "extensor response" is to the most superficial observation, not an isolated phenomenon, however lightly obtained. The muscles activated are the flexors only and the response never involves the extensors of the limb. There is usually no active rebound, but the momentary diminution of tone that follows a reflex may allow the limb to extend passively with gravity. There is no crossed extension reflex with its plantarflexion of foot and toes.

In cases of extreme spasticity in flexion, we occasionally see a crossed flexion reflex, synchronous with—or slightly later than—the homolateral flexion reflex. There is no doubt that, in cases with diminished or absent extensor reflex activity and increased flexor reflex irritability, a crossed symmetrical reflex flexion may occur. It is, however, exceptional, though it accounts for a small percentage of cases of so-called crossed "extensor response."

Intermediate between the two groups of spastic paralysis is a class of progressive spastic paraplegias, from compression or intramedullary lesions of various kinds, in which we are able to see a transition from paraplegia in extension to paraplegia in flexion.

This change is only found in increasingly extensive transverse lesions, i.e., it depends on more and more complete severance of cord and brain. It is characterized, as I have already pointed out, by *progressive disappearance of reflex activity in the extensor group of muscles.* As this change goes on, the flexion reflex tends to become more facile and forcible. It no longer has to overcome a maintained reflex tonus in its antagonists.

A final condition is that of "spinal shock." This is seen in cases of complete transverse lesion of the cord, and in coma preceding death. Case 1 in Appendix is an example of the former.

Lewandowsky and Neuhof [27], and Dejerine and Valensi [16], have reported cases of complete cord lesion, verified by autopsy, in which signs of reflex activity were present in the legs.

In Lewandowsky's case, an "extensor response" was obtained, and also "reflex contractions of the thigh muscles." Following prolonged faradism an ankle-jerk was also transiently obtained.

In Dejerine and Valensi's case, pinching of the skin of the thigh gave a brisk flexion at hip and knee, but for mechanical reasons no "extensor response." In both cases there was complete flaccid palsy with sensory loss and absent tendon-jerks.

In three cases of my series there have been, according to Bastian's [9] law, complete transverse lesions. One case is given at length (Case 1, Appendix). All showed the presence of reflex activity in the flexors, and in these muscles alone. This was, it is true, minimal, and reflex flexion could only be obtained from the distal extremity of the limb. In each case the minimal response was a hamstring contraction. In one case, at onset, no toe movement was obtained, only flexion at hip and knee. All were characterized, as were the cases quoted above, by complete absence of any signs of reflex activity in the extensors. This fact is most significant. In the higher animals, e.g., monkey, recovery from "spinal shock" is late and very incomplete, occurring much later and less completely in extensors than in flexors. In man, shock is more profound, but, for a period at least, there is evidence of reflex activity in one group of muscles, the flexors, until "isolation dystrophy" destroys all possibility of spinal cord function.

(*b*) *Sensory Disturbances in the Limb.*

The interruption of central afferent paths has no effect on the flexion reflex. As typical a flexion reflex is seen in cases with complete as in those with no sensory loss.

In a case of lesion of the lower dorsal cord of sudden onset following parturition, and probably thrombotic in origin, there was complete motor paralysis and sensory loss in the lower limbs. Bedsores rapidly developed and very shortly the legs went into flexion, and the typical clinical picture of paraplegia in flexion, as described by Babinski, resulted. There was a very facile and powerful flexion reflex and "extensor plantar response." Within two months the sensory disturbance cleared up entirely and at present sensation is normal. The motor paralysis and attitude of flexion of the limbs remain almost unchanged, as does also the flexion reflex. It is in cases of paraplegia in flexion that the flexion reflex is best developed. Such cases are generally indicative of severe involvement of the cord, either by compression or in advanced intramedullary lesions, e.g., transverse myelitis, disseminated sclerosis. Very commonly profound sensory loss is present and it is in these cases that we see the reflex movements to best advantage, since all voluntary effort and control are absent, and the patient is unaware both of stimulus and response.

It may be said, therefore, that even profound interruption of central afferent paths has no modifying effect on the flexion reflex.

As regards the crossed extension reflex, it is not possible to make so absolute a statement. As already pointed out, this reflex depends for its occurrence on the presence of a reflexly active extensor group of muscles. Now, in cases of complete or extreme sensory loss, it is not the rule to find this condition, rather do we find the more advanced condition of paraplegia in flexion in which no crossed extension reflex occurs. I conclude that the integrity of at least some afferent paths to the brain is essential for the presence of reflex activity of the extensors.

Any interruption, however, of the afferent side of the spinal reflex arc at the appropriate level, if sufficiently complete, will cause the disappearance of all reflex activity.

Chapter IV.—Summary of Clinical Findings.

(1) In spastic paralysis of the lower limbs the most common reflex movement is that of flexion. This consists, when complete, of flexion at the hip and knee with dorsiflexion of the foot and toes, more especially of the hallux. I speak of this as the flexion reflex of the lower limb. It occurs as the apparently spontaneous flexor spasms so prominent in many cases of spastic paraplegia, and on appropriate stimulation of the limb.

(2) The reflexogenous area or "receptive field" of the reflex comprises the skin and deep structures of almost the entire limb. The threshold of stimulation is lowest on the sole, especially over the outer half.

In general, stimuli of "nocuous" or harmful character are most adequate to produce a reflex, but, on the sole, light painless pressure applied by stroking will evoke a reflex response.

From the proximal fourth of the thigh, reflex extension is sometimes obtained. This is infrequent, and apart from the crossed extension accessory to the flexion reflex, reflex movements of extension hardly ever occur.

(3) The "extensor" type of plantar response never occurs without evidence of reflex contraction of more proximally situated flexor muscles, notably the hamstrings. And conversely, a flexion reflex always includes the dorsiflexion of the hallux, known as the "extensor response." Both are elicited in the same manner and from the same receptive field. They are inseparable and are clearly parts of the same phenomenon.

(4) Inhibition of the extensors of the limb—the antagonists of the contracting muscles—is seen in the flexion reflex as inhibition of the knee-jerk and knee-clonus, and as diminution of the hypertonus of the extensors while active flexion lasts; following this, "extensor rebound" may be seen.

(5) When the limbs are spastic in extension, a crossed extension reflex may occur on eliciting a flexion reflex. Of this crossed extension reflex, plantarflexion of the foot and toes is a part, and constitutes the "crossed plantar reflex" of Babinski and others. Both flexion and crossed extension reflexes consist in a single movement of the limb of the same duration as the exciting stimulus, and showing no rhythmic character.

(6) The occurrence of a crossed extension reflex depends on the existence of a high degree of reflex activity in the extensors, such as is found in the affected limb in hemiplegia and in "paraplegia in extension." It never occurs apart from this.

(7) The flexion reflex is seen at its highest development in the condition of "paraplegia in flexion," in which, the limb extensors having lost their reflex activity, that of the flexors, which is phasic in type, has reached this extreme form, where it constitutes the condition in question.

(8) The two clinical forms of spastic paralysis of the legs, first clearly described by Babinski, are found to have this essential difference: that

while the extended form shows reflex activity in both flexor and extensor groups of muscles, the flexors alone retain this in the flexed form. In paraplegia in flexion the knee- and ankle-jerks may be absent, but the hamstring-jerks are always brisk. The statement, therefore, that the tendon-jerks may be absent in this type of spastic paralysis is not correct. They still exist in the only muscles with reflex action, namely, the flexors.

(9) Comparison of the two forms shows that the specific reflex action of the extensors consists in the maintenance of a high degree of tone in these muscles, "*a static reflex*." The flexors, on the other hand, under the same conditions of pyramidal tract affection, develop *a reflex activity "phasic" in type*, giving rise to reflex movement.

Chapter V.—Physiological Considerations.

§ 1.—*The Physiological Significance of the Flexion Reflex and of the "Extensor Response."*

It is abundantly clear that the reflex phenomena occurring in the lower limbs, as an accompaniment of pyramidal tract affection, present the closest possible analogies with those described by Sherrington in his animal experiments. We must now consider whether they are capable of the same explanation, and whether we are justified in applying to man the conclusions at which Sherrington has arrived as the result of his physiological researches on animals.

The flexion reflex, including the dorsiflexion of the foot and hallux, which has been shown to occur in man, on the application of appropriate stimuli to the limb, and the crossed extension reflex which may accompany it, are strictly analogous with the flexion and crossed extension reflexes of the hind limb of the spinal and decerebrate animals, in regard to the nature of the stimulus, the extent of receptive field and the form of the motor response, in each instance.

Sherrington concludes that this reflex is protective in function. It differs from "spinal stepping" in that it is a single and not an alternating reflex, and that it is elicited by a totally different form of stimulus. Nocuous stimuli applied to the limb produce a single flexion, i.e., a flexion lasting with the stimulus, and never an alternating flexion and extension. "Spinal stepping," on the other hand, is characteristically alternating and rhythmic, and is elicited by passive movement of the limb—never by cutaneous nocuous stimuli applied to it.

Is the reflex obtained clinically in man, then, of the nature of a

protective reflex, Babinski's "Réflexe de défense," or does it partake of the nature of "spinal stepping"?

Marie and Foix [30, 31] are of opinion that the reflex is analogous with "spinal stepping," and has no protective significance. They point out that the movement obtaining in it is unvarying in form, whatever the site of the stimulus, and shows no trace of adaptation to circumstances. For, although the movement is one of withdrawal when the sole is stimulated, the same movement in response to stimulation of the thigh constitutes an actual approach to the site of injury. The reflex cannot, therefore, they say, be protective.

However, the fact cannot be overlooked that when the reflex activity of the limb is at its lowest, as in "spinal shock," and when the causative lesion is confined to the pyramidal tract, as in hemiplegia, the receptive field of the reflex is minimal, and in these circumstances is limited to the sole, or even to a part of the sole. Further, the threshold of stimulation is always lowest on the sole, and no matter what the extent of the receptive field, a response is always to be obtained here. The sole is, therefore, the most essential part of the receptive field; and a reflex, with the characteristics I have described, elicited most readily from the sole by stimuli of nocuous character, may surely be regarded, legitimately, as protective in function.

A more important objection to the opinion held by these authors, however, is that it does not seem to recognize the essential differences between the nociceptive flexion reflex of Sherrington and "spinal stepping," differences which completely distinguish the two reflexes and prove the identity of the one we are considering with the flexion reflex of the hind limb of the spinal animal. The alternating and rhythmic movement of the locomotive reflex of the spinal animal is obtainable by passive extension of the flexed limb; i.e., by proprioceptive stimuli, or by nocuous cutaneous stimuli applied to the opposite limb or the skin of the perineum, i.e., remote from the reacting limb, while the single movement of the flexion reflex is elicited by nocuous stimulation of the reacting limb, and never by crossed or proprioceptive stimuli.

As to the relationship between the flexion of the limb and the "extensor plantar response," various views are held by different authors. Marie and Foix [31] and Noica [33] believe that the two phenomena are part of a general flexion of the limb. According to these authors the "extensor response" is the minimal manifestation of a reflex automatic movement of general flexion, which itself is but a part of the complex motor mechanism of progression. That it is part of the flexion reflex

my observations have clearly shown, but analysis of the reflex in varying conditions of development has demonstrated, in addition, that it is not the minimal motor response, this being found in the inner hamstrings, as Sherrington has also found to be the case in the flexion reflex of the spinal animal. Marie and Foix have recorded a further interesting point, which I am able to confirm, that when the reflex is elicited by stimulation of the proximal part of the receptive field, the "extensor response" is less ample and may even fail. This is the phenomenon referred to by Sherrington as "local sign," and which I have mentioned already.

Babinski, by whom the "extensor response" was first described under the name of "le phénomène des orteils," while regarding the limb flexion as defensive in nature, considers the "extensor response" to be a distinct and separate reflex, a thing apart, for which he offers no explanation, and which remains for him a mystic and inexplicable sign of pyramidal tract affection.

In support of his view he instances cases in which reflex flexion of the limb was accompanied by plantarflexion of the hallux, i.e., by a "flexor response." But I have shown, and figs. 3 and 4 record, that where actual plantarflexion does accompany flexion at hip and knee, the two phenomena are not synchronous, and that a dorsiflexion of foot and hallux, that is, an "extensor response," does occur as part of the reflex flexion.

Babinski also refers to a case recorded by Dejerine and Valensi [16], and verified at autopsy by Dejerine and Long [17], in which, following total transverse lesion of the cord, there was a strong reflex flexion to be obtained on pinching the skin of the thigh. On stimulating the sole, however, a plantarflexion of the hallux resulted. From this, both Babinski and Dejerine conclude that the two phenomena are separate and distinct.

Reference to the original clinical description of the case in the *Revue Neurologique* reveals the following statement: "Les pieds sont en varus-équin et les orteils en flexion. Cette déformation est aujourdhui fixée par des rétractions fibro-musculaires. . . . Le réflexe cutané plantaire se fait en flexion et il est plus fort à droite." It is difficult to see how any other movement than plantarflexion could occur in the circumstances, and the observation and inference are alike valueless.

Finally Babinski [6] has shown that the application of an Esmarch's bandage to the spastic limb causes a transient conversion of the "extensor response" to a plantar response of normal "flexor"

type, which at once gives place to complete absence of plantar reflex, although at the same time the defence reflexes are greatly increased in intensity. Since the two phenomena are so differently affected by this procedure, he infers that they must be separable and distinct.

It can be demonstrated, however, as Marie and Foix have shown, that this experiment is quite illusory. The application of the bandage is such as to render ischæmic the muscles involved in the "extensor response," but not those causing flexion at hip and knee, while at the same time it acts as a strong stimulus to reflex flexion, in which, naturally, the ischæmic muscles take no part. On the first application of the bandage, the "extensor response" becomes "flexor" in type and then all plantar response disappears. This transient conversion, Marie and Foix suggest, is due to the fact that the more superficial anterior tibial group of muscles, subserving the "extensor response," are rendered bloodless earlier than the deep plantar muscles responsible for the plantarflexion.

There is nothing, therefore, in these arguments brought forward by Babinski to invalidate the view held here as to the nature of the "extensor response," which my observations show to be part of the flexion reflex of the limb. It is always present in the fully developed reflex, and conversely, when it occurs it is invariably accompanied by reflex contraction of the proximal flexors of the leg, notably of the inner hamstrings, which are the motor focus of the reflex.

Van Woerkom [43] is another author for whom the "extensor plantar response" is a reflex in itself. To account for it, he advances a hypothesis, which may be briefly described as based on the supposition that primitively the hallux is a prehensile organ with movements peculiar to itself, to which it reverts in condition of pyramidal tract involvement. This view is interesting, but must remain purely speculative, and is certainly superfluous.

I conclude, therefore, not only that the flexion reflex of the lower limb, obtained as here described, is a reflex, protective in function and strictly analogous with the nociceptive flexion reflex of the hind limb of the spinal (or decerebrate) animal, but also that the "extensor response" is an integral part of this reflex.

The "extensor response" becomes, according to this hypothesis, part of a co-ordinated, purposive movement complex, and it is interesting to consider how it compares in this respect with the normal type of plantar reflex—the "flexor response."

The relationship of the two phenomena has been the subject of much

discussion. Babinski, in dealing with the "skin reflexes," observes that whereas the abdominal reflexes disappear in pyramidal lesions, the plantar reflex, which is regarded as belonging to the same class of reflex, undergoes a qualitative change. V. Gehuchten [41] divides skin reflexes into normal and pathological. The former disappear in lesions of the pyramidal tract, the latter only appear in these circumstances. He supposes, then, that the normal plantar reflex has gone, and has been replaced by a new reflex.

According to Marie and Foix, and in this respect my observations would tend to confirm their view, the normal plantar response is a unisegmental reflex strictly comparable with the abdominal and cremasteric reflexes, and involving no co-ordination whatever, that is, no reciprocal innervation; it disappears in pyramidal lesions, being replaced by a new reflex, which is not purely cutaneous, and which is a co-ordinated movement, namely, the flexion reflex. In the normal plantar reflex we see on stimulation of the first sacral segmental skin area, a simple contraction of muscles innervated from this segment: the short flexors of the digits and tensor fasciæ femoris.

I have described the variability of the contractions of the adventitious muscles in the "flexor response," and the participation in the movement of muscles belonging to both flexor and extensor groups. I would further draw attention to the extremely limited receptive field of the normal reflex and to the fact that it is indeed a cutaneous reflex pure and simple. It differs fundamentally, therefore, from the pathological plantar response.

In addition to the flexion reflex, I have described a crossed extension reflex, and I have shown that the crossed plantar reflex bears the same relation to this that the "extensor response" bears to the flexion reflex; namely, that the foot and toe movement in each case does not constitute the whole reflex movement, but merely the most readily apparent part. In the case of the crossed extension reflex, therefore, the full extent of the movement is only seen if the reacting limb is held passively flexed while stimulating the crossed sole. If this be not done, a plantarflexion of the foot and toes is the only visible response.

Contrary to what has been assumed, this crossed plantar reflex differs from the normal "flexor response"; and I have described how this difference in form may best be seen.

Complete analysis of the crossed extension reflex shows that it bears the closest possible analogy with the crossed extension reflex of Sherrington's animals, in regard to the character of the exciting

stimulus and the extent of the receptive field, the form of the response, and the circumstances under which it occurs. Obtained as a reflex accessory to the flexion reflex it has this further interest, that it affords convincing evidence that the processes of reflex action and motor innervation in man show no essential or qualitative differences from these physiological processes in animals, and I propose to develop this analogy still further in the succeeding section.

In the meantime, a reference is necessary to the occasional occurrence of a "crossed extensor response," i.e., a "Babinski response" obtained on stimulation of the opposite sole.[1] This is only seen under certain specific conditions, in my experience. In the extended form of spastic paralysis of the lower limb, homolateral flexion is always accompanied by crossed extension, when any crossed reflex is seen, and this connotes crossed plantarflexion of foot and toes.

However, where the reflex activity of the extensors is relatively less than that of the flexors—namely, in paraplegia in flexion, and in conditions transitional between this and the extended form of spastic paralysis—we do see a crossed flexion reflex with its integral "extensor response." In such cases occur bilateral spontaneous flexor spasms. Sherrington has noted, in certain parallel circumstances, the occurrence of crossed reflex flexion. In man, therefore, as in animals, there is this occasional exception to a general rule laid down by Sherrington, that in the hind limbs of the spinal animal simultaneous reflex movements are opposite in phase in the two limbs.

The phenomenon of "flexor rebound" following crossed extension is, however, as I have shown (*see* figs. 8 and 9) responsible for a certain proportion of "crossed extensor responses."

[1] In view of the fact that the "extensor response" is part of a general movement of flexion, while the toe movement in the normal plantar response, the "flexor response," is, physiologically considered, a movement of extension, it is unfortunate that the accepted terminology is the direct converse of the facts. The expressions "extensor response" and "flexor response" are widely used by neurologists in this country, and I have therefore been compelled to make use of them, though it is very difficult to prevent confusion in the mind of the reader when speaking of a "crossed extension reflex" and of a "crossed extensor response"; two very different phenomena involving antagonistic groups of muscles.

I wish, therefore, to emphasize that these empirical and misleading names are used here in their accepted sense, and have no physiological connotation such as the terms "flexion reflex" and "crossed extension reflex" have. I have consequently placed them in inverted commas to avoid as far as possible all confusion.

In spite of the obvious objections arising from the practice of applying the name of the discoverer to any clinical phenomenon—and the literature on the subject of the reflexes provides many striking examples of this abuse—it seems almost better to speak of the "Babinski response" or of "Babinski's toe phenomenon" rather than to employ terms having a false physiological significance. This is more especially so in this instance, in view of the inevitable confusion that must follow any attempt to correct the present terminology.

§ 2.—*The Dual Nature of the Motor Innervation of the Limb.*

My observations have further established a point of great significance in the study of spastic paralysis, and one not hitherto appreciated; namely, the relationship between the two clinical forms of this condition, in the lower limbs.

It is not my purpose in this paper to deal extensively with the question of contracture except in so far as it concerns the reflex movements of the limb. The two phenomena are intimately connected, as I have already shown in the case of paraplegia in flexion, a condition in which the extensor muscles show diminished or absent reflex activity, while their antagonists, the flexors, show a high degree of reflex activity.

The complete differentiation of the reflex action of the musculature into two systems by the processes of disease, as seen in this form of spastic paralysis of the lower limbs, affords a striking proof of the existence of two systems of motor innervation in man, which are strictly comparable with those differentiated in animals in Sherrington's researches.

I have, in an earlier part of this paper, briefly alluded to this double motor innervation of the musculature of the limb in animals, and a more detailed reference is necessary.

Sherrington has shown that in the innervation of the musculature there are two great reflex systems, each employing in a characteristic manner its own group of muscles.

One system uses the extensor muscles and maintains in them a steady tonus which enables the decerebrate animal to stand, and which Sherrington [38] regards as an expression of reflex posture. The central nervous mechanism of this reflex must be situated in the brain-stem, since it is found only in the decerebrate animal. After transection of the cord at, or below, the caudal end of the pons, the reflex tone of the extensors—the "decerebrate rigidity"—disappears, and a condition of "spinal shock" ensues. This affects only the previously tonic muscles, the extensors, the flexors showing a slight and transient depression.

The other reflex system employs the flexor muscles of the limbs, and its reactions are characteristically phasic, that is, they consist of reflex movements. This reflex is purely spinal, since it is unaffected by severance of cord from brain. Certain of the reactions of this system are protective in function, for example, the flexion reflex of the hind limb. Reflexes of this type are characteristically elicited by nocuous stimuli; they are the least affected by "spinal shock" of all reflex

reactions, and emerge earliest from it. They are "prepotent": that is, they can replace reflexes of other forms, and most easily the tonic postural reflex of their antagonists, the extensors.

In the decerebrate animal both these reflex systems are found in a state of uncontrolled activity. The limb extensors show a maintained tonus—a static reflex; while, by appropriate stimulation of the limb, reflex movements of the flexors can also be obtained, which break through and temporarily replace the tonic postural reflex. While active flexion is in progress the extensors are inhibited, but resume activity with a momentary increase when flexion ceases. This "post-inhibitory exaltation" of activity is known as "extensor rebound."

The question of the actual identity of the "prespinal centre" for the extensor reflex mechanism will at once be raised. It must be admitted that the evidence of its existence is purely physiological. Yet if this be sufficiently great, we are justified in speaking of such a centre, although its morphological identity may be uncertain.

The physiological proofs can be summarized as follows: An animal whose brain-stem has been transected at the level of the anterior corpora quadrigemina, exhibits a state of active muscle-tone in a physiological muscle group, and in this group alone. Section of the afferent nerve of a muscle in this group abolishes its tone. The reflex arises, therefore, in the muscle itself. Section of the posterior columns of the cord does not abolish the reflex, but section of one lateral column abolishes it below the level of section. The central paths of the reflex are, therefore, appear to lie in the lateral columns. More precise details of their identity are wanting. A second transection of the central nervous system, at any level below the caudal end of the pons, completely abolishes the reflex below this level. The grey matter, then, of this reflex arc lies at a "prespinal" level. Thiele [40] has shown that the actual level is that of the plane of the paracerebellar nuclei, and with Horsley and Clarke [14] has demonstrated these nuclei to be the source of the "decerebrate rigidity" of Sherrington, that is, of the "cerebellar influx" of Jackson. Sherrington also finds that ablation of the cerebellum itself does not abolish the rigidity [36].

There is, therefore, clear evidence of the existence of a reflex centre placed within a certain area of the brain-stem, limited anteriorly by the anterior limits of the mid-brain, and posteriorly by the level of the plane of the hinder end of the pons. It is not in the cerebellum.

As yet this has not been finally identified with any specific mass of grey matter in the brain-stem. Nevertheless, there is adequate

evidence of the existence of this centre governing extensor tonus in the limb musculature, and probably situated in the paracerebellar nuclei.

The identity of the efferent path from this reflex centre, Sherrington does not specify further than to say it is in the lateral column, *but it cannot be the pyramidal tract, since this has already been interrupted by the decerebration. It must, therefore, be an extrapyramidal motor path.* The significance of this will be seen later.

In the purely spinal animal, the reflex activity of the flexor muscles is as great as in the decerebrate, indeed the threshold of stimulation of the flexion reflex is even lower. Such activity as the extensors now show is secondary to flexor reflexes, and only occurs in protective reflexes or in spinal stepping, as a phase in an alternating movement of the limb. The flexors, therefore, have their centre in the spinal grey matter. In each case the integrity of an afferent path from the muscle to the reflex centre is essential, and section of this, or "de-afferentation" of the muscle, abolishes all reflex activity in that muscle.

Sherrington [36] sums up the matter as follows: "Two separable systems of motor innervation appear thus controlling two sets of musculature; one system exhibits those transient phases of heightened reaction which constitute reflex movements; the other maintains that steady tonic response which supplies the muscular tension necessary to *attitude*. . . . These two systems, the tonic and the phasic reflex systems, co-operate, exerting influences complemental to each other upon various units of the musculature."

To what extent are we justified in applying these conclusions to the elucidation of the phenomenon of spastic paralysis in man?

Clinically, we are not concerned with actual decerebration, but in hemiplegia we have a condition amounting to unilateral decerebration as far as the motor innervation of the limb is concerned.

In hemiplegia the function of the great corticospinal motor path is partly, or completely, abrogated and on analysis we find that the condition resulting in the lower limb presents the closest possible analogies with that of the hind limb of the decerebrate animal.

In hemiplegia, and in any spastic paralysis due to a lesion confined to the pyramidal system, we find a spastic paralysis of the limb varying in intensity, but absolutely constant in type.

In a typical hemiplegia, or lateral sclerosis of the cord, the lower limb lies fully extended with the foot plantarflexed. The limb extensors are very rigid and present brisk tendon-jerks, namely knee-jerk, ankle-jerk, and possibly clonus. This group of muscles also retains relatively

more voluntary power than its antagonists, the flexors, which muscles show less of the spasticity characteristic of the extensors; for example, passive flexion of such a limb often requires considerable force, but once flexed there is no muscular resistance to passive extension.

Wernicke and Mann [29] first recorded both this selective distribution and inverse relationship of weakness and spasticity.

In addition to the spasticity of the limb we note in the flexor group of muscles a brisk tendon-jerk in semimembranosus and semitendinosus, and the presence of reflex movements of flexion with the "extensor response."

That is, both extensor and flexor groups of muscles show a condition of increased and involuntary reflex activity, manifested as hypertonus in the extensors and as reflex movement in the flexors.

This clinical picture, which Babinski [2] calls that of spastic paralysis "en extension," and German authors, "Streckkontractur," is characteristic of a cerebral lesion, or of any lesion, spinal or cerebral, limited to the pyramidal tract. As regards spinal lesions, Babinski [2] considers that it is in systematic lesions of the pyramidal tract in the cord that the condition characteristically occurs, while Oppenheim notes the extreme rarity of flexion contracture of the legs in amyotrophic lateral sclerosis and other pure pyramidal tract lesions, and says that it only occurs as a terminal phenomenon.

All authors are agreed that in hemiplegia the extensor form of spasticity is constant. The fact is so significant that I think it cannot be too well emphasized. Lewandowsky [26] speaking of hemiplegia says, "im allgemeinen die typischen Verteilung der Kontractur auf die Beuger am Arm und die Strecker am Bein zustande kommt." Oppenheim [34] is equally explicit: he says, "Beugekontraktur des Beines kommt nur selten und wohl nur unter ungewöhnlichen Bedingungen vor." V. Monakow [32] also emphasizes this point: he says, "Flexionskontraktur (Verkürzungskontraktur) der unteren Extremitäten cerebralen Ursprungs (nach Hemiplegie und als Spätkontraktur) ist sehr selten; sie kommt nur dann vor, wenn ernstliche nutritive, die passive Verkürzung fordernde Störungen in der Muskeln selbst vorhanden sind, oder wenn zu der gewöhnlichen hemiplegischen Kontraktur noch protrahirte Muskelkrämpfe (corticale event. mesencephalen Ursprungs) hinzutreten."

The cases recorded of flexion contracture of the leg in hemiplegia are exceedingly rare. Devic and Gallavardin [18] have described such an instance, in which the lesion was a hæmorrhage in the internal

capsule. The affected leg was the seat of strong flexor spasms which, from the description given, appear similar to those familiar in paraplegia in flexion. The state of the deep reflexes in the limb is not stated, and it is not possible to say whether the two conditions are really comparable.

In the patients I have examined in the National Hospital and in a large series of old-standing hemiplegias which I have had the opportunity of seeing in the St. Marylebone Infirmary, I have not met with flexion contracture in the leg. Nor have I ever seen it in spinal cord lesions, where involvement of structures other than the pyramidal tract could be excluded. In amyotrophic lateral sclerosis, paraplegia in extension is the rule, and only very rarely in the terminal stages of the condition does flexion contracture occur. The *degree* of pyramidal involvement has no influence on the *form* of spasticity in the leg, and *we may conclude, therefore, that a pyramidal lesion, when uncomplicated, gives rise always to the extended form of spastic paralysis in the lower limb.*

Horsley [24] has pointed out the identity of the "decerebrate rigidity" of animals with the rigidity of the leg in hemiplegia, and if in the latter condition we analyse the state of the two muscle groups in the lower limb, the extensors and the flexors, we find the most striking resemblance to "decerebrate rigidity." In both instances we see an extensor group of muscles showing a maintained hypertonus—a "static reflex," and a flexor group showing, as its characteristic form of activity, movement—a "phasic reflex," but not rigidity such as is found in the extensors.

In connexion with this question of a specific type of reflex activity in each of the two muscle-groups, it is not, of course, suggested that the extensors show no phasic activity, or that their antagonists the flexors, have no residual tonus. The presence of alternating movements of the limbs, involving reflex extension as a phase, negatives such a suggestion. Also, the presence of brisk hamstring-jerks, in all cases of spastic paralysis, indicates that the reflex activity of the flexors is not absolutely phasic, but rather shows, to use Sherrington's phrase, "phases of heightened activity." It is clear, however, that in spastic conditions of the lower limbs, the activity characteristic of the extensor muscles is in the form of "tonus," while that of the flexors is phasic, and consists of movement.

We know from the work of Sherrington that decerebrate rigidity is a reflex phenomenon depending on the integrity of a reflex arc passing

from muscle to reflex centre in the brain-stem and thence to the muscle again. We also know that the afferent and efferent links in the arc lie in the lateral columns of the cord. I have pointed out that, since decerebration has interrupted the pyramidal tract, the efferent path for the tonic impulses evoking the characteristic rigidity in the extensors must be an extra-pyramidal one.

Spinal transection severs this path, with the result that the extensors lose their tone, and henceforth show practically no trace of reflex activity. The flexors, on the other hand, which have never been the seat of maintained hypertonus, continue to show unimpaired reflex activity of the specific phasic type. It is thus evident that the efferent path conveying tonic impulses to the extensors has no physiological connexion with the flexors, the reflex action of which is, therefore, seen to be purely spinal.

It becomes of interest to see whether in man, in the parallel condition of spastic paralysis in extension, we have any evidence of the existence of a special extrapyramidal motor path determining in the extensors of the leg the tonic rigidity which is seen to occur, and which, as has been pointed out, so closely resembles the decerebrate rigidity of animals. If this be so, it should be possible to recognize a type of spastic paralysis analogous with the condition found in the spinal animal, in which the flexors alone retain reflex action. Such a phenomenon would be evidence of the existence of a motor innervation of the extensors, other than that depending on the integrity of the pyramidal tract.

In certain severe spinal lesions we do, as I have shown, actually find a type of spastic paralysis, which on analysis is seen to reproduce minutely the state of affairs found in the spinal animal.

This is the condition described by Babinski as paraplegia in flexion. Babinski has found that this clinical type of spastic paralysis occurs commonly in severe diffuse spinal lesions. That it is never seen in cerebral lesions, I have already observed. It is well known, and my observations have confirmed this, that in progressive paraplegia due to compression or to intramedullary disease, the legs pass from the extended to the flexed condition, and it is generally recognized that paraplegia in flexion indicates very severe involvement of the cord.

As described by Babinski [7] it is characterized by the following features:—

(1) Muscular rigidity in flexion, which is intermittent and thus gives rise to involuntary movements of alternating flexion and extension, the former predominating.

(2) Profound loss of voluntary power.

(3) Diminution or absence of the tendon-jerks.

(4) Exaggeration of the "defence reflexes" and an "extensor response."

Moreover, he says, in typical examples, examination of the cord reveals relatively slight secondary degeneration of the pyramidal tracts. On account of the absence of the tendon-jerks, which is in striking contrast to their activity in the extended form of spastic paralysis, he calls this type of spastic paralysis, "cutanéo-réflexe," and the other "tendino-réflexe." This classification is misleading, and loses sight of the essential difference between the two conditions.

An analysis of the state of reflex activity of the two muscle groups in the limb—namely, the extensors and flexors—reveals the significant fact that the extensors show little or no reflex action, while the flexors show a high degree of reflex activity.

In the extensors we find no tone, the knee- and ankle-jerks are greatly diminished or actually absent, and there is no clonus.

In the flexors we find an extreme degree of reflex activity; the leg is flexed, and it requires considerable force to overcome the spasm, which, however, is notably intermittent, and has not the tonic character seen in the extensors in the extended form of spastic paralysis. Spontaneously, or on the lightest cutaneous or deep stimulus to the limb, strong flexor spasms occur; each of these being, as I have shown, a complete flexion reflex. Percussion of the tendons of the hamstrings gives invariably a brisk tendon-jerk, a significant fact in view of Babinski's description of the condition.

The essential feature of this form of spastic paralysis is that in it only the flexors retain any reflex activity, the extensors showing little or none. Whereas a lesion of the pyramidal tract alone produces a state of increased activity of both flexor and extensor muscles in the limb, the lesion in these cases has in addition destroyed all signs of reflex action in one group of muscles, leaving that of the other great group unaffected, and even increased since it is no longer antagonized. The two reflex systems are *differently* and *separately* affected. The analogy between this condition and that of the spinal animal is at once apparent.

A consideration of the character of the lesions giving rise to this condition shows that they are of several clinical and pathological types, but they have in common their severity, the fact that they are spinal in localization, and that they interrupt, more or less completely, conduction between the cord and the brain.

Short of actual total transverse lesion, the more complete the separation between ventral horn cells and brain, the more highly developed is this form of spastic paralysis.

The point raised by Babinski that there may be little or no descending pyramidal degeneration on pathological examination of the cord in such cases, has no special significance, for, as Gordon Holmes [23] has shown, there is no constant direct relation between the severity of the clinical manifestations and the secondary descending degeneration. A focal lesion of the cord may cause extreme spastic paralysis and loss of sphincter control, and yet, on autopsy there may be no descending pyramidal degeneration.

Since a lesion confined to the pyramidal tract never produces paralysis in flexion, we may assume that, as in the spinal animal, the interruption of an extrapyramidal path is an essential condition of its development. The fact that this form of spastic paralysis is seen in spinal lesions at the highest levels proves that this motor path must arise at a higher level than the cord, while its non-occurrence in cerebral lesions proves, similarly, that its origin must be at a lower level than that of the fore-brain. We are compelled, therefore, to assume the existence of an efferent motor path, other than the pyramidal tract, passing to the extensor muscles, or rather to their ventral horn cells, and to these alone.

It will be seen that the analogy between the state of reflex activity in the hind limb of the "spinal animal," and that of the lower limb of man in paraplegia in flexion, is as close as that between "decerebrate rigidity" and paraplegia in extension.

It would appear, therefore, that, as in animals, we have in man two separate systems of innervation of the limb musculature, each employing a definite group of muscles and giving rise in each of these to a specific form of activity, tonic in the one, clonic, or phasic, in the other.

The extensors need, for the maintenance of their hypertonus, the integrity of a reflex arc passing from the muscles themselves to a reflex centre in the grey matter of the brain-stem, probably the paracerebellar nuclei. The efferent limb of the arc consists of an extrapyramidal tract passing thence to the ventral horn cells of the extensor muscles.

The reflex activity of the flexors, on the other hand, is unaffected by the interruption of this path to their antagonists, and we must conclude that their reflex arc is purely spinal.

Recent investigations tend more and more to emphasize the important part played by extrapyramidal motor paths.

Foerster [22], v. Monakow [32], and Rothmann [35] attribute some of the phenomena of spastic paralysis to the taking over of the functions of the impaired pyramidal system by phylogenetically older motor systems, such as the rubro-spinal; and more recently the valuable work of Kinnier Wilson [42] has shown, in the most striking manner, the physiological possibilities of these paths.

While, however, the view attributing important functions to motor paths other than the pyramidal system is one widely held and strongly supported by clinical and pathological evidence, it has not been previously suggested that such a motor system may show a selective innervation of one group alone of the limb muscles.

Yet, Jackson's idea of the motor innervation of muscles "having the most tonic, the most confluent movements" by a special nervous mechanism differing from that innervating those muscles having "the most changing movements" is a distinct foreshadowing of the conditions of reflex action found to obtain in animals by Sherrington, and not less of the parallel state of affairs my observations show to exist in man.

The view advanced here of the tonic innervation of the extensor group of muscles in the lower limb by an extrapyramidal projection system, in addition to its cortico-spinal innervation by the pyramidal system, the flexors of the limb having an innervation by this latter system alone, harmonizes with the speculations of Jackson and experimental findings of Sherrington.

The conception of the lower motor neurone (the "final common path" of Sherrington) receiving impulses from more than one efferent projection system ("internuncial common paths") has been developed by Sherrington, and the double innervation of the extensor muscles of the lower limb provides a striking example of this factor in the co-ordination of reflex action.

Another phenomenon, which I have observed in a case of disseminated sclerosis, points to the existence of a grouping of the nervous representation of the limb muscles into extensor and flexor in the pyramidal tract itself.

The case was in every respect typical, and in history the patient stated that his right leg was much weaker than the left. Examination revealed the following condition: Both legs lay extended and adducted; passive movement revealed considerable spasticity in the extensors of both limbs, equal on the two sides. In power the extensors of both sides showed a slight and equal defect. The flexors, on the other hand, were very unequally affected. On the right they were extremely weak

and very little voluntary power of dorsiflexion of the foot or toes was present. On the left side flexion and dorsiflexion were normal. The tendon-jerks on both sides were brisk, and ankle-clonus was present.

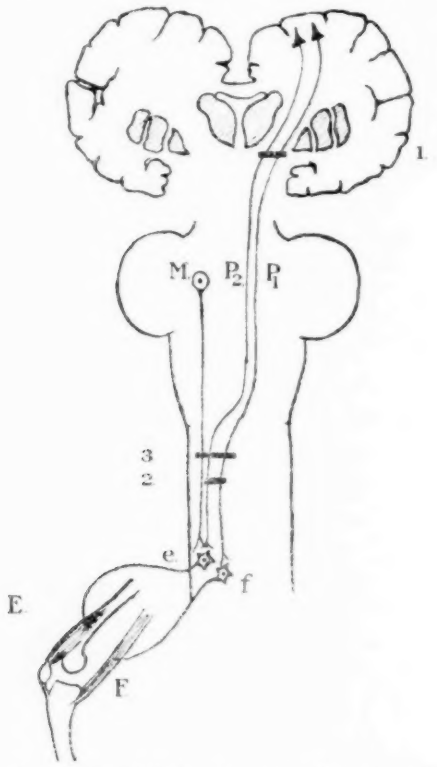

FIG. 12.—In this diagram P_1 and P_2 are pyramidal fibres going to *e* and *f*, the ventral horn cells of extensor and flexor muscles respectively. E and F represent these muscles. M is the extra-pyramidal path arising probably in connexion with the paracerebellar nuclei and going to the extensor neurone.

A lesion, 1, gives rise to a paralysis of the extended type. Reflex activity of both groups of muscles is increased.

A lesion, 2, a lateral sclerosis, produces the same result. Both involve the pyramidal system alone.

A lesion, 3, besides interrupting the pyramidal tract, involves the extrapyramidal path also, and in such a case paralysis in flexion results.

The plantar response on the right side was "extensor," on the left a normal flexor was obtained and no trace of a pathological plantar reflex was to be found, nor any reflex flexion of the limb.

According to my view, pyramidal fibres to the flexors and extensors in the right leg were equally involved. On the left side, on the other hand, pyramidal fibres to the flexors were intact, only those to the extensors being involved. Hence, on this side there was a normal plantar response.

Examples of spastic legs with increased jerks, clonus, and a normal plantar response, are familiar to all neurologists, and would repay closer observation from this point of view.

Current theories do not allow the existence of any grouping of fibres in the pyramidal tract — either physiological or anatomical. However, the observation above recorded points to a physiological grouping, and further investigations are necessary to elucidate this question.

The foregoing diagram represents the hypothesis of the motor innervation of the limbs, which I put forward (fig. 12).

§ 3.— *Total Lesions of the Cord.*

In comparing the condition of paraplegia in flexion with that of the hind limbs in the spinal animal, it is necessary to bear in mind the clinical picture of complete transverse spinal cord lesion in man.

As Bastian [9] first pointed out, in this condition there is complete flaccid paralysis with absence of the tendon-jerks, and complete sensory loss. There are also sphincter defects, and rapid wasting and degeneration of the muscles sets in. Occasionally, according to Collier [15], certain skin reflexes are present in reduced degree, and constitute the sole evidence of self action in the limb.

This clinical picture is often seen transiently following some severe and acute cord lesion, such as occurs in fracture dislocation of the spine or in acute transverse myelitis. In such cases partial recovery ensues, but before this appears we can regard the condition as one of complete physiological transverse lesion of the cord.

Therefore, although the condition of paraplegia in flexion may present many points in common with the condition of the hind limbs of the spinal animal, it is clear that it differs widely from the clinical picture presented by the leg in complete cord lesions in man, i.e., in the "spinal" man. This difference, however, is only one of degree, as a study of recorded cases and my own observations show.

Sherrington has pointed out that following spinal transection, a condition of "spinal shock" ensues. This affects mainly the extensors,

and the flexors but slightly and transiently. Yet while the rabbit recovers almost completely from spinal shock in a few hours, the dog recovers more slowly and less completely, while higher animals, like the monkey, only show long delayed and partial recovery. In the spinal rabbit, a knee-jerk reappears in a few hours, while in the monkey it may not be elicitable for months. Sherrington considers this shock to be due to the rupture of certain efferent paths arising in the brain-stem and also to the phenomenon of "isolation dystrophy": that is, to trophic degenerative changes in the nervous elements when cut off from efferent impulses from the brain.

Man differs from the lower animals only in that, in him, spinal shock is more intense, and that the phenomena of shock are perpetuated by the occurrence of actual degenerative changes in the nervous elements themselves, which naturally destroy all function in the isolated cord.

Moreover, Sherrington has observed that protective reflexes, or as he calls them "nociceptive reflexes" are relatively little depressed by spinal shock, and are the reflex phenomena which emerge earliest from this condition. The flexion reflex of the hind limb affords an excellent example of this.

He says: "In the monkey and in man spinal shock is not only peculiarly intense but peculiarly long lasting. The withdrawal from the isolated cord of influences it is wont to receive from centres further headward may induce an alteration of trophic character in spinal cells—an 'isolation dystrophy'—visible, it may be, as Nissl's chromatolysis.

"This 'isolation dystrophy' ensuing on shock would add itself as a longer lasting, in some elements, perhaps, a permanent, depression. . . . . The deeper depression of reaction into which the higher animals as contrasted with the lower sink when made 'spinal' appears to be significant of this, that in the higher types, more than in the lower, the great cerebral senses actuate the motor organs and impel the motions of the individual."

When we come to analyse the "skin reflexes," which are preserved in cases of complete transverse cord lesions, we find them to consist in minimal forms of the flexion reflex, which I have described. In other words, there is retained in a depressed form the great protective or "nociceptive" reflex of the limb. It is further to be noted that this reflex involves the activity of flexor muscles only.

A case of complete traumatic division of the spinal cord by a bullet

wound in the dorsal region is recorded by Lewandowsky and Neuhof [27]. It bears out the view I have advanced.

For a period of several days following the lesion, before degenerative changes had set in in the muscles, an "extensor response" was obtained together with "reflex contractions of certain of the thigh muscles."

These reflex phenomena were increased in facility and intensity by prolonged faradization of the limb, and a transient ankle-jerk but never a knee-jerk, was also obtainable by these means. However, the most active and enduring reflex seen was the Babinski plantar response. This establishes the occurrence of a flexion reflex in the "spinal" man.

I have personally observed three cases of physiologically complete cord lesion, one within twenty-four hours of a fracture dislocation of the dorsal spine. The legs were flaccid, powerless, and absolutely insensitive, all tendon-jerks were absent and there was retention of urine. Stroking of the sole gave as response a visible contraction of the hamstring and of tensor fasciæ femoris. Twenty-four hours later a dorsiflexion of foot and feeble flexion of the knee and hip were obtained, and on some occasions an actual "extensor response" was obtained, but tired readily. At the moment of writing—six days after the trauma—the condition present is unchanged, but muscular wasting has appeared.

Another case seen seven days after the onset of an acute transverse myelitis gave an identical result. This case is recorded at length in the Appendix, and affords a striking example of the progressive evolution of reflex activity in a spinal cord recovering from physiologically complete section.

Even accepting the presence of a flexion reflex in the lower limbs of the "spinal" man, it has to be admitted that the condition of flaccid paralysis with depressed reflex activity differs widely from that of paraplegia in flexion. However, the difference is merely one of degree, in that in the latter the flexors show not a minimal but a maximal amount of reflex activity. In no other respect is there any essential difference. Still, how is this difference to be explained?

Dejerine [17] refuses to regard the flexion reflex as evidence of spinal function liberated from cerebral control, as Marie and Foix suggest when they call the phenomenon a "Réflexe d'automatisme médullaire," because, he says, it only occurs in incomplete destruction of the cord, and never when transection is absolute. This, as I have shown, is not strictly true. It is true, however, that these reflex phenomena are most marked when the separation of cord from brain is incomplete. To this we may add that the more profound the interruption is, short of

complete section, the more marked are these reflexes. The condition of paraplegia in flexion is evidence of this.

It must be admitted that in the condition of disease in which we have to study these reflexes in man the lesions are brutal and incomplete, and we seldom have exact knowledge of their extent.

I would suggest that in the condition of paraplegia in flexion we have the "spinal" man without the phenomena of "spinal shock" or "isolation dystrophy." It is the final separation of cord from brain that induces these phenomena and depresses at first, and finally destroys the reflex activity of the flexors.

In paraplegia in flexion, according to this view, there is complete liberation of spinal reflex function without that depression of reaction and subsequent destruction of nervous elements which anatomically complete division produces.

The presence of a very depressed, minimal flexion reflex during the short period before degenerative changes ensue in nerve and muscle, in cases of complete cord transection in man, is definite evidence that there is no essential qualitative distinction between the condition of paraplegia in flexion and that of the flaccid paralysis seen in the latter circumstances, and strongly supports the view that these two clinical pictures differ merely in the presence in one of them of "spinal shock."

To sum up, we may say that there is strong evidence that the condition of paraplegia in extension, including here that of the lower limb in hemiplegia, is analogous with the "decerebrate rigidity" of animals deprived of their hemispheres: that paraplegia in flexion and the flaccid paralysis of complete cord transection in man differ only in degree, and are analogous with the condition of the hind limb of the "spinal animal."

In paraplegia in extension both extensor and flexor groups of muscles show a high degree of involuntary reflex activity, and this is specific in character for each group.

In paraplegia in flexion and in the flaccid paralysis of the complete cord lesion the flexor muscles alone show any sign of reflex action.

Since these two reflex mechanisms can be thus separately affected, they must be differently represented in the central nervous system: that is, there are two systems of motor innervation of the limb musculature.

In any lesion of and confined to the pyramidal system, we find both these reflex systems constantly active, i.e., we see the extended form of spastic paralysis of the leg, and never the form with contracture in flexion. A lesion producing this is always spinal, and must involve the interrup-

tion of some efferent path in the cord, other than the pyramidal tract, since this is already involved; I say efferent paths, since the condition of paraplegia in flexion may be found with an intact afferent system.

This further involvement of an extrapyramidal path affects only the extensors, and integrity of this path appears to be essential if these muscles are to display any reflex action. The centre from which it arises is, I conclude, analogous with that "prespinal centre" for the extensor muscles in animals for the existence of which Sherrington has brought forward convincing evidence.

I hope to demonstrate, in a subsequent paper, that the phenomena of spasticity and contracture, which have been the subject of much recent work by Foerster, are also capable of complete explanation in accordance with the hypothesis here advanced.

Chapter VI.—General Summary.

(1) The reflex movements of the lower limbs in spastic paralysis are almost entirely movements of flexion of the limb. With flexion is included dorsiflexion of foot and toes, more especially the hallux.

These movements occur, apparently spontaneously, as the well-known flexor spasms seen in certain cases of paraplegia, and also on appropriate stimulation of the limb. This reflex I have called the flexion reflex of the lower limb.

(2) The "receptive field" of this reflex commonly includes the whole limb, skin and deep structures, except for a small portion of the proximal part. The reflex is not purely cutaneous. The form of stimulus most effective is that having a nocuous or harmful character, but light painless stimuli applied to the distal extremity of the limb, the sole, suffice to induce the reflex. The muscles actuated are the flexors and dorsiflexors of the limb, and the reflex response is a simple movement of flexion at all the joints.

(3) Of this reflex flexion, dorsiflexion of the hallux is an integral and inseparable part, and this constitutes the "extensor" type of plantar response, the Babinski toe phenomenon, the appearance of which is recognized to be pathognomonic of pyramidal tract affection.

The "extensor response" is, therefore, part of the flexion reflex, and analysis proves it to be an integral part of this, never occurring without reflex contraction of the proximal limb flexors.

(4) The physiological significance of the flexion reflex is that of a defensive reflex, and it has no relation to "spinal stepping."

(5) Concurrently with contraction of the limb flexors in this reflex is inhibition of their antagonists, and all the phenomena of "reciprocal innervation" are seen in the reflex.

(6) Accessory to this reflex is reflex extension of the crossed limb which, following Sherrington, I call the crossed extension reflex. This includes plantarflexion of foot and toes, "the crossed plantar reflex" of many authors. This is shown to differ from the normal flexor plantar reflex.

(7) The study of spastic paralysis of the lower limbs, from the point of view of reflex movement, confirms the existence of two clinical types. These have been described by Babinski as "paraplegia in extension" and "paraplegia in flexion."

I have pointed out here that the essential difference between the two types is that, while in the extended form of spastic paralysis of the lower limbs both extensor and flexor groups of muscles are in a state of heightened reflex activity in the flexed spastic leg, only the flexors retain this, the extensors showing diminished or absent reflex action.

(8) The reflex activity of these two groups of muscles is, it is here put forward, of specific type in each case. The extensors show a maintained tonic reflex—the spasticity of the extended leg. This may be called a static reflex. The flexors show, characteristically, reflex action of phasic type, which gives rise to reflex movements.

(9) In a cerebral lesion or in a purely pyramidal tract affection, however complete, we see constantly the extended type of spastic paralysis in the legs.

The type of spastic paralysis in flexion, or, as it has with scant appreciation been called, "spastic paralysis with flexion contracture," is only seen in spinal cord affections; most commonly in severe, diffuse lesions, never in pure lateral sclerosis involving only the pyramidal tracts.

To produce this condition the interruption of an efferent tract in the cord other than the pyramidal tract is essential. This path arises caudal to the fore-brain and probably in the pons, in connexion with the paracerebellar nuclei.

The effect of a lesion of this extrapyramidal motor path is to abolish the reflex tone in the extensor muscles, without affecting the reflex activity of the flexor muscles.

(10) The condition of paraplegia in flexion is an example of unantagonized flexor reflex action, and it shows intermission, due to the phasic nature of this, until permanent contracture ensues.

Since the condition is found in the severest spinal lesions, the reflex

activity of the flexors may be considered purely spinal in origin and not dependent on any efferent path from the brain.

(11) In complete spinal transection, physiological or anatomical, the reflex activity of the limb is almost completely absent. But a minimal flexion reflex persists on stimulation of the sole, and thus affords further evidence of the purely spinal nature of this reflex. The extensors show no reflex activity in these cases.

(12) The condition of flaccid paralysis of the limbs in complete lesions of the spinal cord differs, therefore, in degree only from that of paraplegia in flexion, which I conclude is analogous to the condition of the hind limbs of the "spinal animal." In complete spinal lesion the phenomena of "spinal shock" and "isolation dystrophy" mask and finally abolish the reflex activity of the flexors.

(13) It will be seen from this that there is a double system of motor innervation of the lower limbs, such as was first suggested by Jackson and verified experimentally in animals by Sherrington.

I have endeavoured to show further that the two factors in this are, first, the pyramidal system which innervates both limb flexors and extensors. A lesion of this path liberates both reflex systems from control, and in the limb extensors we see the extended form of spastic paralysis.

The second component is a tonic innervation along an extrapyramidal tract arising in the brain-stem and passing to the extensor muscles only. Interruption of this abolishes the reflex activity of these muscles, leaving that of the flexors unimpaired. The condition of spastic paralysis in flexion then ensues, the reflex action of the flexors being purely spinal.

(14) An analysis of the phenomenon of spastic paralysis of the limb extensors in man demonstrates the close analogy existing between the physiological principles of motor innervation in animals and in man.

APPENDIX I.

Illustrative Cases.

Case 1.—*An instance of physiologically complete cord lesion. Flexion reflex present in minimal form, no other reflexes present. Slow recovery during which the legs passed from complete flaccid paralysis through a condition of paraplegia in flexion to that of paraplegia in extension, with corresponding changes in the reflexes.*

F. T., a girl, aged 19, was admitted into the National Hospital under the care of Dr. Batten on February 2, 1914. Eight days before admission she began to develop a rapidly progressive paraplegia, which became complete on the

second day. For six days previous to examination, there had been complete flaccid paralysis with sensory loss and retention of urine.

Condition on February 2. There was absolute flaccid paralysis of both lower limbs, which lay extended and rotated out. They were absolutely devoid of tone. On attempting to sit up in bed the umbilicus moved up. There was absolute loss to all forms of sensibility below a line drawn round the trunk one inch above the umbilicus. There was retention of urine. All tendon-jerks were absent. The faradic excitability of the muscles was good, though they were readily fatigued.

The Flexion Reflex.—Light stroking of the sole, but no form of stimulation of any other part of the limb, gave a quick contraction of the hamstrings and of the anterior tibial muscles. There was slight flexion of the knee and hip without movement of the toes, but a feeble dorsiflexion of the foot. If the stimulus to the sole was very light it was possible to get a motor response limited to a palpable contraction of semitendinosus and semimembranosus, but too weak to produce any movement of the limb. Whenever the reflex contraction appeared in the muscles distal to the knee, it was well marked in all the proximal flexors. That is, the minimal response was in the proximal muscles. Pin-prick on the sole gave similar results.

Strong faradism applied to the muscles directly by means of needle electrodes for periods of from ten to thirty minutes had no effect either on the flexion reflex or the tendon-jerks.

On February 4. No change, except that to intense stimulation of the sole the small toes dorsiflexed feebly, but there was no movement of the great toe. No further change till February 8. The limbs now showed general wasting, and faradic excitability was somewhat reduced.

February 10. All the tendon-jerks were absent, but percussion over the heads of the metatarsals gave reflex flexion of the limb. The flexion reflex was facile from the sole. The minimal response was contraction of the hamstrings and of tensor fasciæ femoris. To stronger stroking of the sole there was added to flexion at hip and knee dorsiflexion of ankle and of all the toes; a good "extensor response." If the dorsiflexion of the foot was resisted the "extensor response" was more ample. The receptive field was still confined to the sole for all forms of stimuli. Strong faradism for a period of thirty minutes was devoid of result.

February 12. Light stroking of the sole now gave a typical "extensor response," accompanied in every case by visible limb flexion. Successive stimulation soon tired the reflex, the response disappearing first distally—i.e., in the toe, and then in the ankle. So that finally a stimulus previously able to evoke a complete reflex now only gave flexion at hip and knee. Oppenheim's procedure for the first time gave a feeble contraction of the proximal flexors, but no toe movement. The motor and sensory condition of the limbs had not changed, and the tendon-jerks were absent.

February 13. The flexion reflex as before. The knee- and ankle-jerks were absent, but now percussion of the tendons of semitendinosus and semimembranosus gave a feeble, palpable contraction of these muscles.

February 17. Knee- and ankle-jerks absent, but hamstring-jerks brisk on both sides. The flexion reflex could now be elicited in complete form by light stroking of the sole, and by pin-prick of the skin up to the level of the calf. Also strong percussion of the shaft of the tibia and of the bony points round the ankle gave a flexion reflex complete in form. The minimal response was still in the hamstrings.

February 22. The tendon of biceps femoris now gave a jerk. Oppenheim's method also gave an "extensor response." The first sign of returning sensibility was seen in slight perception of the vibrations of a tuning-fork placed on the tibia. A complete flaccid paralysis remained.

At this time, twenty-six days from the date when the lesion became complete, the flexors showed rapidly returning reflex activity, but the extensors were still absolutely without any such activity. Flexor reflex activity was present from the first, when the lesion was physiologically complete.

March 8. Flexion reflex now obtainable by pin-prick of any part of the limb surface below the middle of the thigh. There was no trace of any crossed reflex response. *The knee-jerks were feebly present for the first time.* These appeared after some sensory return indicated that the lesion was no longer complete. Ankle jerks absent.

March 16. Knee- and hamstring-jerks present, ankle-jerks still absent. The flexion reflex was gaining in force and amplitude, and the receptive field was increasing in extent. Pin-prick of the lower part of the abdominal wall gave feeble reflex flexion, in which a toe movement was just perceptible. The "extensor response" was very ample and facile on light stimulation of the sole, but could not be obtained without contraction of the proximal flexors of the limb. Complete flaccidity remained.

During April practically no change occurred.

May 11 (thirteen weeks from onset). The legs lay half flexed, and when straightened slowly returned to this position, and there was now slight but definite tone in the hamstring muscles and in the hip flexors. Less definite hypertonus was felt in the knee extensors. Any sudden passive movement of the limbs caused a strong flexor spasm.

During May, June and July, strong flexor spasms began to appear, and the legs, although not powerfully flexed, always lay fully flexed, and sudden passive extension increased the spasm. The knee-jerk was still of poor facility, but the ankle-jerk did not appear. The flexion reflex was very facile to all forms of stimulation.

In August strong adductor spasm began to appear, and more evidence of spasticity in the extensors.

During September the flexor spasms abated notably and the patient began to remark that sudden extensor spasms came on and made the limbs very rigid in extension. Their attitude was now always one of extension.

In October, the ankle-jerk at last reappeared. There was now strong extensor rigidity and flexor spasms were occasional. The flexion reflex was as facile as ever, but the flexion was not so ample and a quick extensor rebound cut it short always.

In September the obturator nerves were crushed to overcome the adductor spasm. This it did completely.

Present condition (October 30).—The legs are very spastic in extension. All the tendon-jerks are brisk and there is ankle-clonus on both sides. There is feeble power of extension of the limb, but as yet scarcely power of flexion, except at the hip.

The flexion reflex is elicitable over a wide field and is ample and complete. The minimal response remains in the hamstrings and in tensor fasciæ femoris, but to any but the slightest stimulus a complete flexion reflex is obtained.

The return of sensation has been much more complete than that of power, and sensory loss is now only moderately marked to any form of sensibility.

This case is instructive from many points of view. It is of special interest here as showing the evolution of the reflex activity of the limb in the process of recovery from "spinal shock" in the first place and also in the clearing up of the lesion, which remained complete for some time according to accepted standards.

The order of appearance of the tendon-jerks is also of the greatest importance. They are seen in the hamstrings; the flexors, almost three weeks before their reappearance in the extensors. Further, in view of the work of Collier [15] and of Lewandowsky and Neuhof [27] on the effect of strong faradism on the tendon-jerks in cases of complete section of the cord, it is of interest to note that in this case frequent faradism appeared to have no effect in accelerating the appearance of the tendon-jerks.

Summarized, we have here a case of temporary physiological solution of continuity of the spinal cord, in which recovery has slowly and partially ensued. While the lesion was total and "shock" was profound, the flexion reflex was still obtainable from some of the muscles, in which it is present when complete, on stimulation of a small receptive field at the apex of the limb, the minimal motor response being in the muscles at the limb base. When fully developed the flexion reflex included flexion at hip and knee and dorsiflexion of ankle and of hallux—the "extensor" type of plantar reflex. This was inseparable from the flexion reflex; it was elicited by the same stimuli, but was evidently not the minimal response. *Pari passu* with the spread of reflex motor response the receptive field also spread until there was obtained from the whole surface of the limb and from most of its deeper structures, the same complete flexion reflex, in all respects identical with the nociceptive flexion of the hind limb of the spinal dog, described by Sherrington.

Further, this reflex response from the limb flexors was obtained

when the lesion was complete, at a stage when no reflex activity was seen in the extensors, and before the presence of the knee-jerk gave any evidence of reflex tone in the extensors. This only appeared after slight sensory return showed that the cord below the lesion was no longer completely separated from the higher centres.

CASE 2.—*An instance of progressive paraplegia passing from the extended condition to that of paraplegia in flexion, the limbs finally becoming flaccid. The dissolution of reflex activity in the lower limbs is seen. The case shows very well the differential effect of a progressive lesion of the cord on the reflex activity of extensor and flexor groups of muscles respectively.*

Percy G., aged 16, was admitted into the National Hospital, in April, 1914, under the care of Dr. Gordon Holmes. He gave a history typical of disseminated sclerosis: Diplopia, misty vision, unsteadiness of limbs and sphincter disturbances, dating from October, 1910. He was previously in hospital in October, 1911, when the diagnosis was made.

He remained fairly well until February of this year, when he had in rapid succession a series of epileptiform fits with vomiting, followed by progressive weakness of the limbs and other disturbances of the nervous system.

On examination in April, he was emotional and fatuous. He had diplopia and nystagmus with left-sided facial weakness. Speech was syllabic and slow. The upper limbs were spastic and unsteady, the left showed some loss of the sense of position.

The lower limbs lay extended and adducted; both showed moderate extensor spasticity, but power was almost completely absent. There was no sensory loss. The tendon-jerks were all brisk, patellar- and ankle-clonus were present at times. Percussion of the sole gave a knee-jerk (Loewy and Cohn).

A facile flexion reflex was present and could be obtained from the whole limb and from the perinæum.

Light stroking of the sole and dorsum of the foot gave a complete flexion reflex. The minimal stimulus gave a visible flexion of the limb, and an "extensor response" could not be obtained without this contraction of the proximal limb flexors. Pin-prick over the whole limb gave a complete reflex. Squeezing of the calf or thigh muscles gave a good reflex.

From whatever part of the limb obtained, and by whatever form of stimulus, the reflex response was unvarying limb flexion with dorsiflexion of foot and hallux.

Accompanying the flexion reflex was a crossed extension reflex. If the crossed limb were previously flexed, this consisted of extension at hip and knee, with plantarflexion of foot and all the toes. The movement was quick and forcible. If the crossed limb lay fully extended the crossed reflex consisted of plantarflexion of the foot and toes (see figs. 8A and 8B).

The reflex flexion of the homolateral limb was soon cut short by a quick extensor rebound, while the crossed extension showed a flexor rebound (fig. 8C). The graphic record shows all these phenomena (fig. 9).

This condition remained more or less unchanged until May 5, when the right leg was noticed to lie often in an attitude of flexion, to which it soon returned when passively extended.

Examination now revealed the following condition. Voluntary power was gone on both sides. The right leg was flaccid, but showed possibly slight tone in the hamstrings. The left leg was much as on admission.

The right knee- and ankle-jerk were absent, those on the left were brisk. The hamstring-jerks were brisk on both sides, especially on the left. The flexion reflex was facile on both sides and had not changed in any way, except that the right no longer showed any extensor rebound, while the left flexion reflex was not accompanied by any crossed extension. The right flexion reflex was so accompanied.

In other words, the extensor group of muscles of the right leg had lost their reflex activity, while that of the flexors was not changed.

However it was repeatedly noticed on this and the few following days that if, while percussing the right patellar tendon, the left flexion reflex were strongly elicited, a feeble knee-jerk could be obtained. *The left flexion reflex was reinforcing the extensors of the opposite limb.*

May 7. The right leg now lay continually flexed and the reflexes were as before. The left leg was still rigid in extension and the reflexes unchanged. The hamstrings of the right leg were now developing a condition of tone upon which frequent flexor spasms were superimposed, and any sudden passive extension of the limb provoked a strong increase of the flexor spasm, including an "extensor response." The limb was also wasting generally, more than the left.

Crossed extension of the left limb persisted till the end of May, when the left limb also went into flexion and lost its knee- and ankle-jerks. Crossed extension of this limb on stimulating the right sole now disappeared finally.

The hamstring-jerks on both sides remained brisk.

Both limbs had now lost the reflex activity of their extensor group of muscles, and the condition of paraplegia in flexion was present. The flexor spasms of both legs were frequent and the flexion reflex was very facile, but the minimal response was now seen to be in the hamstring muscles on light stimulation of the sole. At first the minimal response had consisted in a complete reflex.

From this date the reflex activity of the limbs declined rapidly, until finally only a feeble flexion could be obtained from the sole, and from no other part of the limb. The minimal response was seen very clearly in the proximal limb flexors and shortly before death stimulation of the sole evoked only this response.

Autopsy confirmed the diagnosis and the cord showed almost complete sclerosis in the dorsal region, only a small area in one ventrolateral column staining by the Weigert method.

This case shows the phenomena observed in Case 1, but inversely. It confirms the views formed as to the nature of the flexion reflex from

the study of that case. It shows the decline of the reflex activity of the extensor muscles of the limb at a time when that of the flexors was unaffected, and affords strong evidence of the existence of two systems of motor innervation of the limb musculature.

APPENDIX II.

Graphic Methods.

The method employed was the direct recording on a revolving drum of the limb movements. In cases where these were of too great amplitude, the thread from the limb passed to the outer circumference of a light multiple wheel such as is used in the kymographs of the physiological laboratory. From a smaller circumference on the same wheel a thread passed to a recording lever.

The graphic recording of the reflex movements of the spastic limbs offers considerable difficulties. The sudden occurrence of strong flexor spasms continually pulls the recording lever off the surface of the revolving drum and even breaks the connecting thread. In many cases repeated efforts were necessary before a presentable record could be obtained. In the course of making records from such cases, one was struck by the continually changing degree of the reflex activity of the spastic limbs. Particularly did this seem the case with the extensor muscles. The hypertonus, the briskness of the tendon-jerks, and the occurrence of clonus altered from moment to moment. The flexion reflex showed much less variation.

To record movements of the whole limb or of the hallux alone, a thread is attached to the tip of the great toe by a piece of adhesive strapping. To record limb flexion apart from toe movement the writing lever is connected with the internal malleolus in the same manner. Either direct or proportional recording can be used.

A metronome connected in circuit with an electric signal is used to record time in seconds.

A record of the time relations of the knee-jerk or of the plantar stimulation in the flexion reflex can be obtained very simply, and does not necessitate the elaborate apparatus described in "Lewandowsky's Handbuch" and elsewhere.

A piece of metallic gauze, such as is used in electro-therapy, is wound round the knee over the patellar tendon. A similar piece is wound round the percussion hammer. Wires lead from each to a

circuit in which are a battery and an electric signal. On striking the patellar tendon, contact is at once made and recorded by the signal on the smoked surface of the kymograph.

In the accompanying diagram (fig. 13) the two alternative methods of direct (D) and proportional (E) recording of limb movement are indicated.

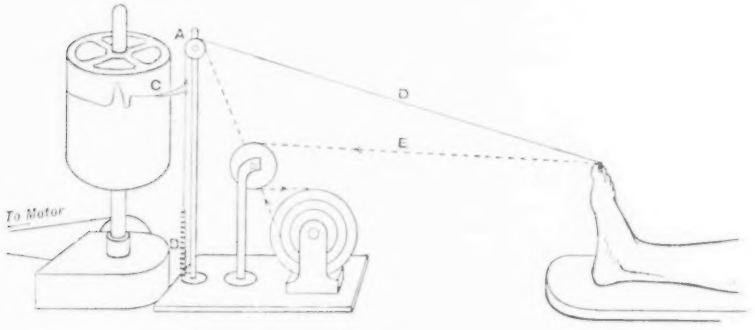

FIG. 13.

The thread in each case passes over a wheel, A, and is fastened to a spring, B. This keeps the thread taut and prevents it from becoming loose in the sudden flexion or extension movements of the limb. The recording lever, C, is fastened to the thread, and consists of a piece of parchment paper.

The drum is revolved at given speeds from a small motor.

The electric signal is not represented.

REFERENCES.

[1] BABINSKI. "Du phénomène des orteils et de sa valeur sémiologique," *Semaine Médicale*, 1898, p. 321.

[2] *Idem.* "Sur une forme de paraplégie spasmodique consécutive à une lésion organique et sans dégénération du système pyramidal," *Bulletins, etc., des Hôpitaux de Paris*, 1899, p. 342.

[3] *Idem.* "Sur la transformation du régime des réflexes cutanés dans les affections du système pyramidal," *Rev. Neurol.*, 1904, No. 1, p. 58.

[4] *Idem.* "Sur le réflexe cutané plantaire," *ibid.*, 1907, No. 14, p. 755.

[5] *Idem.* "Paraplégie spasmodique organique avec contracture en flexion et contractions musculaires involontaires," *ibid.*, 1911, No. 2, p. 132.

[6] *Idem.* "Modifications des réflexes cutanés sous l'influence de la compression par la bande d'Esmarch," *ibid.*, 1911, vol. ii, p. 951.

[7] *Idem.* "Contracture tendino-réflexe et contracture cutanéo-réflexe," *ibid.*, 1912, No. 14, p. 77.

[8] Babinski. "Réflexes tendineux et réflexes osseux," Paris, 1912, p. 64 *seq.* Reprinted from *Bulletin Médical*, October—November, 1912.

[9] Bastian. "On the Symptomatology of Total Transverse Lesions of the Spinal Cord, with Special Reference to the Condition of the Various Reflexes," *Med. Chir. Trans.*, 1890, vol. lxxii, p. 151.

[10] Beevor. "Ueber die Koordination und Repräsentation der Muskelbewegungen im zentralen Nervensystem," *Ergebnisse der Physiologie*, 1909, 8 Jahrgang, S. 326.

[11] Brown-Séquard. "Sur l'arrêt immédiat de convulsions violentes par l'influence de l'irritation de quelques nerfs sensitifs," *Archives de Physiologie*, 1868, vol. i, p. 157.

[12] Byrom Bramwell. "Note on the Crossed Plantar Reflex," *Rev. Neurol. and Psychiat.*, 1911, vol. ix, p. 49.

[13] Charcot. "Lectures on Diseases of the Nervous System," New Sydenham Society's Translation, 1881, vol. xc, p. 93.

[14] Clarke and Horsley. "On the Intrinsic Fibres of the Cerebellum, its Nuclei and its Efferent Tracts," *Brain*, 1905, vol. xxviii, p. 13.

[15] Collier. "The Effects of Total Transverse Lesion of the Spinal Cord," *Brain*, 1904, vol. xxvii, p. 38.

[16] Dejerine et Valensi. "Paraplégie cervicale d'origine traumatique. Ecrasement de la moëlle épinière, etc.," *Rev. Neurol.*, 1911, No. 14, p. 141.

[17] Dejerine et Long. "Examen histologique d'un cas de section complète de la moëlle cervicale inférieure d'origine traumatique," *Rev. Neurol.*, 1912, No. 24, p. 769.

[18] Devic et Gallavardin. "Sur un cas remarquable de contracture post-hemiplégique," *Rev. Neurol.*, 1903, No. 2, p. 53.

[19] Fairbanks. "The Contralateral Plantar Reflex," *Rev. Neurol. and Psychiat.*, 1911, vol. ix, p. 225.

[20] Foerster. "Die Kontracturen bei den Erkrankungen der Pyramidenbahn," Berlin, 1906.

[21] *Idem.* "Relations between Spasticity and Paralysis in Spastic Paralysis," Seventeenth International Congress of Medicine, London, 1913, Section XI, Neuropathology, vol. ii, p. 55.

[22] *Idem.* "Das phylogenetische Moment in der spastischen Lähmung," *Berlin. klin. Wochenschr.*, 1913, Nos. 26 and 27, S. 1255.

[23] Holmes, Gordon. "On the relation between loss of Function and Structural Change in Focal Lesions of the Central Nervous System, with special reference to Secondary Degeneration," *Brain*, 1906, vol. xxix, p. 514.

[24] Horsley. "The Functions of the Cerebellum," *Brain*, 1906, vol. xxix, p. 446.

[25] Hughlings Jackson. "Remarks on Rigidity of Hemiplegia," *Medical Examiner*, April, 1877, p. 271.

[26] Lewandowsky. "Handbuch der Neurologie," Berlin, 1913, Bd. I, t. ii, S. 696.

[27] Lewandowsky und Neuhof. "Über Wiederbelebung der Reflexe nach Rückenmarksverletzung," *Zeit. f. d. gesamte Neurol. und Psych.*, 1912, Bd. xiii, S. 444.

[28] Loewy. "Die plantare Reflexzone für den M. quadriceps," *Neurol. Centralb.*, January, 1913, S. 90.

[29] Mann. "Über den Lähmungstypus bei der cerebralen Hemiplegie," *Volkmann's klinische Vorträge*, 1895, Inn. Med., No. 39, Nos. 30-54, S. 355.

[30] Marie et Foix. "Les réflexes d'automatisme médullaire," *Rev. Neurol.*, 1912, vol. xxiii, p. 657.

[31] *Idem.* "Réflexes d'automatisme médullaire et réflexes dits de défense. Le phénomène des raccoureisseurs," *Semaine Médicale*, October 22, 1913, p. 505.

[32] v. Monakow, "Gehirnpathologie," Wien, 1897.

[33] Noica. "Sur les Réflexes cutanés du dos," *Rev. Neurol.*, 1912, vol. xxiii, p. 134.

[34] Oppenheim. "Lehrbuch der Nervenkrankheiten," Berlin, 1913, 6te Aufl., Bd. ii, S. 896.

[35] Rothmann. "Restitutionsvorgänge bei cerebralen Lähmungen, &c.," *Deutsch. Zeitschr. f. Nervenheilk.*, 1914, Bd. l, S. 406.

[36] SHERRINGTON. "The Integrative Action of the Nervous System," London, 1906.
[37] *Idem.* "Flexion Reflex of the Limb, Crossed Extension Reflex, and Reflex Stepping and Standing," *Journ. of Physiol.*, 1910, vol. xl, p. 28.
[38] *Idem.* "Remarks on the Reflex Mechanism of the Step," *Brain*, 1910, vol. xxxiii, p. 1.
[39] STROHL. "Les réflexes d'automatisme médullaire: Le phénomène de raccourcisseurs," *Thèse de Paris*, 1913.
[40] THIELE. "On the Efferent Relationship of the Optic Thalamus and Deiters' Nucleus to the Spinal Cord, with special reference to the Cerebellar Influx of Hughlings Jackson and the Genesis of the Decerebrate Rigidity of Ord and Sherrington," *Journ. of Physiol.*, 1905, vol. xxxii, p. 358.
[41] VAN GEHUCHTEN. "Réflexes cutanés et Réflexes tendineux," *Rev. Neurol.*, 1900, vol. viii, p. 736.
[42] WILSON, KINNIER. "Progressive Lenticular Degeneration, &c.," *Brain*, 1912, vol. xxxiv, p. 296.
[43] WOERKOM, v. "Sur la signification physiologique des réflexes cutanés des membres inférieurs," *Rev. Neurol.*, 1912, vol. ii, p. 285.

Publications Recently Received.

[Notes on a book under this heading do not preclude a subsequent review.]

Die funktionellen und hirnanatomischen Befunde bei der japanischen Tanzmaus. Von Dr. Taco Kuiper. S. 154, with many plates under separate cover. Rotterdam: Hengel, 1913.

This admirable monograph is founded on the microscopical study of the brains of five fully grown and four newly born dancing mice. After a clear summary of previous work the author gives his own observations. He believes that the movements performed by these extraordinary animals vary greatly; some resemble "forced" movements, some are more like attacks of "whirling," whilst other mice carry out definite dancing evolutions. The animals differ greatly in liveliness and force, and the loss of general muscular tone varies in extent. All his animals were undoubtedly deaf. The lesion is a degeneration of the whole system of the eighth nerve ["Octavus-system"] including both its peripheral and central endings. The plates are excellent.

Les Réflexes d'Automatisme médullaire, le Phénomène des Raccourcisseurs. [Travail du Service et du Laboratoire de M. le Professeur Pierre Marie]. Par Le. Docteur André Strohl. Pp. 164. Paris: Steinheil, 1913.

In this thesis Dr. Strohl set himself to review the path of the different reflexes in man and to investigate in particular that pathological group attributed by Marie and Foix to spinal automatism. He follows these authorities in believing that the knee-jerk is a true reflex, and that, when the cerebrospinal conduction is diminished or destroyed, the cord develops a new activity, if the lesion has not altogether destroyed its functions. The manifestations of this new activity, the so-called "defensive reflexes," form the subject of the second half of this work. He deals in detail with the "triple flexion" reflex, direct reflex extension of the whole limb and the homolateral flexion with contralateral extension evoked by violently bending the great toe downwards. The discussion is accompanied by graphic records of the movements described, taken in cases of spastic paralysis. He accepts the view that the extensor plantar reflex is a part of complete limb flexion, but as

he attributes all these reflexes to forms of "stepping" and denies their defensive origin it follows that the Babinski extension of the great toe is also part of the act of walking. This position is opposed to the view held by Sherrington and his followers.

An Anatomical Guide to Experimental Researches on the Cat's Brain: A series of 35 frontal sections. By Dr. C. WINKLER, Professor of the Municipal University in Amsterdam, and Dr. ADA POTTER. Pp. 133. Amsterdam: Versluys, 1914.

The cat's brain was cut into frontal sections and thirty-five positions have been chosen for illustration. Each plate is accompanied by a complete description and occasionally by accessory figures illustrating special points, as, for instance, the microscopical structure of the cortex cerebri. The object of this work was to provide a descriptive basis for experimentation on the cat's brain, but the plates are so clear and so excellently reproduced that they are in themselves of permanent interest. The generosity of Mr. Laan has not only made it possible to produce this atlas, but has enabled the authorities of the neurological laboratory in Amsterdam to offer it to those institutions engaged in serious neurological work.

Die Halluzinosen der Syphilitiker. Von Privatdozent Dr. FELIX PLAUT. S. 116. Berlin: Springer, 1913.

This forms one of the monographs edited by Alzheimer and Lewandowsky, and is of great interest, firstly, as the latest opinion of Plaut on the condition of the cerebrospinal fluid in various forms of syphilitic affections, and secondly as the record of an ill-understood group of syphilitic manifestations. He believes that the Wassermann reaction is positive in the cerebrospinal fluid from most patients with syphilis, although "a metaluetic process is out of the question." The absence of a positive reaction in the blood gives an 80 per cent. probability against cerebrospinal syphilis. The author then describes at considerable length a number of cases of syphilis of the nervous system which simulated dementia præcox, primary dementia, hallucinatory paranoia and other forms of mental disease. Particular stress is laid on cases where hallucinations were present.

Meningococcus Meningitis. By HENRY HEIMAN, M.D., and SAMUEL FELDSTEIN, M.D. With introduction by HENRY KOPLIK, M.D., all of Mount Sinai Hospital, New York. Pp. 313, with 4 Plates and 31 Figures. Philadelphia: Lippincott, 1913.

This little book is by far the best account we have seen of cerebrospinal meningitis due to the *Meningococcus intracellularis*. Each chapter deals with one aspect of the disease in a succinct, accurate, and at the same time eminently

readable fashion. The history, bacteriology, epidemiology, mode of dissemination, pathological anatomy, and clinical types of the disease are excellently described. Infantile postbasal meningitis is shown to be due to the same cause as the epidemic forms; the condition of the cerebrospinal fluid and the best methods for lumbar puncture and serum infusion into the spinal canal are clearly laid down. The mode of action and composition of the four sera, most commonly employed, are stated, and the clinical results put forward without exaggeration. The chapter on treatment is evidently the fruit of much personal experience.

Beiträge zur Frage nach der Beziehung zwischen klinischem Verlauf und anatomischem Befund bei Nerven- und Geisteskrankheiten. Bearbeitet und herausgegeben von FRANZ NISSL, in Heidelberg. Erster Band, Heft 1. S. 91, mit 34 Figuren. Berlin: Springer, 1913.

Professor Nissl came to the conclusion that there was not sufficient intellectual co-operation between the clinician and the pathologist; each was interested in his own aspect of the case, and neither was competent to appreciate the other's point of view. The pathologist was not capable of carrying out the clinical examination of a patient up to modern requirements, and so lost touch with the clinician, who was equally unable to make a complete histological examination. He therefore selected certain cases, not necessarily rare or peculiar, for complete study by a clinician and a pathologist. Meetings were held at which these results were brought before all the assistants and other workers in the clinic; at the same time the specimens were shown and explained. These combined reports were so successful that he determined to publish them, and this pamphlet, containing a description of three patients, is the first part of vol. i. The first is a case of syphilitic mesaortitis and endarteritis obliterans of the cerebral vessels; the pathological report is excellent, but its interest is greatly enhanced by the complete life-history of this old soldier, work shy, alcoholic, violent and given to petty thieving. The second case is an atypical one of dementia paralytica, and the third a combination of this disease with arterio-sclerosis and gummatous cerebral syphilis. These are just the "common" cases which excite little interest without the close co-operation of the clinician and the pathologist.

Trauma und Nervenkrankheiten. Von PAUL SCHUSTER, in Berlin [Sonderabdruck aus dem Handbuch der Neurologie von LEWANDOWSKY]. S. 128. Berlin: Springer, 1913.

This is a reprint of Dr. Schuster's article in Lewandowsky's Handbook. He points out that the whole aspect of the medical man towards an injury has changed since the introduction of Workmen's Compensation, and the theme of this article is the effect of an accidental injury on a diseased and on a

presumably normal nervous system. He deals with the questions of long deferred apoplexy, the possible traumatic origin of general paralysis, tabes dorsalis, syringomyelia, myelopathies and other forms of organic diseases, and then gives a short account of traumatic hysteria and the neuroses. The article is extremely practical and should be consulted by anyone who expects to be cross-examined on these difficult aspects of injury to the nervous system.

Die Hysterie. Von Professor Dr. M. LEWANDOWSKY, in Berlin [Sonderabdruck aus dem Handbuch der Neurologie von LEWANDOWSKY]. S. 192. Berlin: Springer, 1914.

Like most of the chapters in the Handbook this article is a full and judicial summary of the present state of our knowledge on hysteria. It is clearly arranged, has an excellent index, and is written in an easy and attractive style. The list of authorities at the close is well selected and contains all the important papers.

Les Techniques anatomo-pathologiques du Système nerveux. Par GUSTAVE ROUSSY et JEAN LHERMITTE. Pp. 255. Paris: Masson, 1914.

This is an excellent manual of histological methods applicable to the nervous system. It is clear, practical, and every procedure recommended seems to have been tested by personal experience. The chapters follow the natural order of preparation of tissues, hardening fluids, section cutting and embedding; staining of the cell of the myelin sheath, of the neuroglia, and of the vascular and connective tissues are considered in separate chapters. The various methods for revealing products of degeneration are given in full, and the last three chapters are devoted to the peripheral nerves, the muscles, and the staining of bacteria.

Anatomia Clinica dei Centri Nervosi. By Professor G. MINGAZZINI. Pp. 936. Turin: Unione Tipografico-editrice, 1913.

An admirable text-book of anatomy of the central nervous system directed particularly to elucidate clinical conditions. The figures are clear and well reproduced, and the descriptions excellent. Such conditions as aphasia are illustrated by the lesions discovered in actual cases familiar from the literature of the subject, and everywhere accurate references are given for the views expressed and examples given.